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THE
LARYNGOSCOPE.

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REVIEW OF AVAILABLE LITERATURE ON THE PHARYNX AND PHARYNGEAL SURGERY FOR 1951.*

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ANOMALIES.

Schmidt[1] presents a case of bilaterally elongated styloid processes in a professional singer. The patient had difficulty in reaching and holding high notes and had some pain and discomfort in the throat. The diagnosis was made by palpation and confirmed by Roentgenography. At operation the stylohyoid ligament was found to be ossified; exposure through the tonsillar fossa was obtained, and one inch of the elongated process was removed on each side. Following this procedure the patient's symptoms cleared up.

Schmidt also describes the embryologic development of the styloid process, which is intimately connected with the hyoid bone. The average styloid process is 2.5 cm. long; longer ones are encountered in only 4 per cent of cases. Various diagnostic points are presented, but it is the opinion of the reviewers that intraoral palpation is unexcelled.

Longacre[2] presents a case of an infant three months old with congenital atresia of the fauces, which he believed to be due to failure of rupture of the buccopharyngeal membrane. The defect was corrected surgically, and the child was in excellent health at the time of this report.

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Numerous variations in surgical technique for the correction of atresia of the posterior nares have been tried; most involve some destruction of the soft parts and removal of part of the nasal septum. Beinfield[3] describes a comparatively simple technique which proved effective and nondestructive in one case of unilateral bony atresia of the posterior nares. An incision is made along the floor of the nose and is extended up onto the septum just inside the cut nares. The mucoperiosteum is elevated from the floor of the nose and septum and from the anterior surface of the bony atresia. The bone closing the posterior nares is then removed with a small mastoid gouge. The mucoperiosteum is replaced and a stellate incision made through the nasal and nasopharyngeal mucosa over the posterior choana. A large rubber tube is passed through this opening and maintained in place five days. Beinfield admits that it is too early to evaluate the final result, but he points out that the principle "is sound and should work out well."

A rather complete discussion of atresia of the posterior nares is included.

DISEASES.

Thibaudeau[4] reports an interesting case of atlantoaxial subluxation which was due not to trauma but to an acute pharyngeal infection complicated by catarrhal otitis in a child nine years old. Penicillin therapy cured the torticollis and a Roentgenogram taken seven days after the first film showed normal bony structure and articulations.

Segundo and associates[5] encountered a syndrome characterized by paralysis of the last four cranial nerves described in 1864 by Hughlings Jackson. The most frequent causes of such symptoms are malignancies of the nasopharynx or base of the skull; trauma, particularly military injuries, and occasionally inflammatory infections involving the base of the skull or lateral adenopharyngeal wall. A case is reported in which the last four cranial nerves were paralyzed, and examination of the patient finally established existence of a lateral pharyngeal abscess with extension to the base of the skull.

Administration of penicillin and streptomycin controlled the infectious process and resulted in gradual disappearance of the paralysis.

Barton[6] believes that subacute thyroiditis as a cause of sore throat is often overlooked by the practicing physician. He has noted that the sensation of a lump in the throat is most often due to thyroid disease, although this has not been our experience. A brief discussion of hoarseness in thyroid disease is given.

Cheney[7] has made a study on the prevention and treatment of colds over a period of 20 years in 2,667 University of California students. For convenience the students were divided into three groups of estimated natural resistance to infection. Few conclusions could be drawn from the study. Some patients with severely deviated septums had no colds, whereas others with no mechanical defects had frequent colds. The best results in decreasing the number of colds were obtained by combined medical, allergic, and ear, nose and throat therapy.

In a discussion of the structure and function of the upper respiratory tract and pharynx, Cawthorne[8] shows that the lymphoid elements of Waldeyer's ring serve to filter and sample bacteria and viruses passing over them and that some antibodies may be elaborated in this tissue in defense against infection. The pathogenesis of the common cold is presented. The possible relationship between the development of bulbar poliomyelitis and recent operations on the tonsils and adenoids is still not known; but it is believed that until more is known of a possible relationship, tonsillectomy and adenoidectomy should not be done when there is a high incidence of poliomyelitis in the community.

Brunner,[9] in reporting a most interesting case of eosinophilic granuloma of the mouth, pharynx and nasal passages, expresses the opinion that the lymphatic tissue of the upper respiratory and digestive tracts does not become frequently involved in chronic systemic disease of the hematopoietic apparatus. The opinion is correct if only gross changes of

the lymphatic tissue, such as enlargements or ulcerations, are considered; however, microscopic changes are frequently encountered. Brunner briefly reviews the findings in Hodgkin's disease in the respiratory and digestive tracts and then reports in accurate detail a 12-year observation on a most interesting case in which the symptoms were suggestive but not diagnostic of Hodgkin's disease. The persistent enlargement of various groups of lymph nodes in the mouth, palate, pharynx and nose presented baffling problems. Tissue eosinophilia was present and the eosinophile count in the blood was 67.25 per cent. A thorough discussion of Hodgkin's disease, incorporated in this article, adds to its interest. Because the case would not conform to the pattern of Hodgkin's, a diagnosis of eosinophilic granuloma was finally made.

Asherson[10] discusses the three different syndromes associated with malignancy of the epipharynx and adjacent areas. These are distinguished by the site of origin of the tumor. The first is Trotter's syndrome, which is stated to be an endothelioma of the lateral wall of the nasopharynx beginning submucosally in the sinus of Morgagni. It is manifested by deafness followed by pain and anesthesia in the third division of the Vth nerve. Later, asymmetry of the palate due to infiltration of the palatine muscle occurs. The second is the pterygopalatine fossa syndrome, which is caused by either a metastatic tumor in the pterygopalatine fossa or direct extension into the pterygopalatine fossa from a tumor of the posterior part of the lateral wall of the nose. It is distinguished by primary pain over the second division of the Vth nerve, fullness of the temporal fossa and later by third division pain, deafness and paralysis of the palate. Finally, the Jacod syndrome of the carrefour petrosphenoidale, which produces the symptoms of both Trotter's and pterygopalatine fossa syndromes, is caused by primary malignancy of the middle cranial fossa involving the second and third divisions of the Vth nerve at the foramen rotundum and ovale. The anatomy and symptomatology of these syndromes are described in great detail and illustrative case reports are cited.

Titche[11] reports a case of chronic peripharyngeal tuberculous abscess in a man 32 years old which was secondary to tuberculous adenitis of the anterior cervical lymph nodes. It was successfully treated by aspiration and local and systemic administration of streptomycin and para-aminosalicylic acid.

Keratosis pharyngis has been reported in the past by many different names and most authors believe it is due to fungi of the Leptothrix species; however, Vaheri,[12] who made a rather complete study of 21 cases involving principally the tonsils and the base of the tongue, found Leptothrix in no case. Friedlander's bacillus and a-streptococcus were most frequently noted. In the cases in which tonsillectomy was done cartilage was commonly found in the tonsils. Keratosis pharyngitis is believed to be a slowly progressive, chronic, self-limiting, inflammatory process. Tonsillectomy is advisable when the tonsils are involved.

Shnee[13] writes an all too short article on a tremendously interesting subject because of the bizarre symptoms which challenge and frequently baffle the diagnostic acumen of the clinician. Patients with paresthesia of the pharynx are frequently seen in the office and, according to Schnee, complain of soreness, dryness, burning, choking, tightness, tickling, itching and even mild dysphagia. Frequently no organic cause can be found to account for these symptoms. The diagnosis is made by a process of elimination. Consideration must be given to such possible causes as hysteria, neurosis, anemia, hypochondriasis and the menopause.

Schnee reports a case in which tightness of the throat, a sensation of a lump and difficulty in swallowing were the chief symptoms. The only finding on clinical examination was a tender spot on the right side of the neck above the hyoid bone. Injection of the superior laryngeal nerve on the right side relieved the symptoms for a period of two months. When these recurred, incision in the neck over the tender spot disclosed an enlarged cervical gland pressing on the superior laryngeal nerve. Two years after removal of the gland there was complete relief of symptoms. The report of

this case is considered important because it proves that frequently meticulous examination will indicate a diagnosis in an otherwise obscure condition which, because of lack of obvious disease, is too often called globus hystericus.

After a brief discussion of pharyngeal infections, Folken[14] reports a case in a boy 14 years old, complicated by erosion of the internal jugular vein. Despite vigorous attempts to control bleeding, repeated hemorrhages occurred, which proved fatal.

During the summer of 1949 and 1950, David and associates[15] observed a group of children, mostly under five years of age, in Jacksonville, Fla., who presented a clinical picture of sudden illness with fever, usually of 48 hours' duration, malaise and anorexia. Examination revealed vesicular or aphthous lesions of the soft palate and anterior faucial pillars. The clinical course of the patients and the appearance of the lesions were strikingly uniform. A viral agent pathogenic for suckling mice was isolated from throat secretions of 12 of 13 patients with this disease. This viral agent was shown to be similar, but of a different serologic type, to the virus isolated in 1948 by Daledorf and Sickles and later designated as the Coxsackie virus. Fortunately, the condition was self-limited, seldom lasting longer than two days; however, the ulcers sometimes persisted as long as seven days. The isolation of the Coxsackie virus from all cases shows the existence of a definite relationship with the clinical disease described by David and associates.

DIVERTICULA.

Crile and Robnett[16] report 11 cases of pharyngoesophageal diverticulum treated satisfactorily by simple inversion of the sac. Briefly the technique is as follows: The incision is made in the usual manner. The sac is dissected free up to its neck, a purse string is placed around the neck and the pouch is inverted into the esophagus. An Ewalt tube is passed into the stomach. The overlying muscle is closed. The Ewalt

tube is removed and the wound closed. Tube feeding is not required. The procedure is aseptic and requires short post-operative hospitalization.

Peickoff[17] reports a case of pharyngeal diverticulum in a man who first noticed, five years before, that on swallowing solid foods, something stuck in his throat and caused him to choke and regurgitate undigested food. Similar episodes recurred on unpredictable occasions. Symptoms became more aggravated and soon a peculiar noisy click followed by a gurgle on swallowing developed. So annoying was this to the family that eventually the patient refused to have meals with his family or strangers; however, he considered the pouch to have one advantage: he noted that at cocktail parties the first few swallows of liquor lodged in the pouch, which had become quite large. Later in the evening he noted that he could have an extra drink when he needed it by pressing the pouch with his thumb. A description of the usual technique for diverticulectomy is included.

Hudson[18] states that few surgeons have the opportunity of studying pharyngeal diverticula because of its rarity. All observers, however, are agreed that as the pharyngeal pouch increases in size it tends to obstruct the esophagus by direct pressure when distended with food; this increases the symptoms and causes anxiety and discomfort to the patient with loss of weight. Hudson reports nine cases of pharyngeal diverticulum in which the patients complained of discomfort on swallowing, choking sensations, regurgitation and unpleasant taste in the mouth, together with loss of weight, depending upon severity of symptoms. Hudson considers nocturnal dysphagia with cough of great diagnostic importance. All nine patients were treated by the one-stage operation, the technique of which is described in detail. Hudson believes that the transverse fibres of the cricopharyngeus muscle situated caudal to the pharyngeal pouch is responsible for the formation of the diverticula, and this muscle is divided at the time of operation. He warns against exerting too much traction on the pouch for fear of amputating too much pharyngeal or esophageal mucosa. Good results were obtained

in all except two patients who had to have a second operation because of early recurrence thought to be due to persistence of the transverse fibres of the cricopharyngeus. Hudson, therefore, believes that deliberate division of these fibres will tend to produce better results.

INSTRUMENTS.

Somers[19] describes how a pencil-sized pocket flashlight can be converted into an electrical handle for the standard nasopharyngoscope and points out that the amount of current is just right.

Inspection of the nasopharynx before, during and following adenoidectomy is essential for performance of adequate adenoidectomy, since the form of the nasopharyngeal space varies widely in different individuals. Toward this end, Neivert and Kallen[20] present a simple palate retractor which doubles as a tongue protractor and greatly facilitates direct inspection of the nasopharynx. It consists of a flat piece of metal seven inches long and five-eighths inch wide with one sharply angulated end. The remainder of the strip is gently curved.

IRRADIATION.

For many years hyperplasia of lymphoid tissue in the nasopharynx has been treated by irradiation. In a most informative manner, Garland and associates[21] discuss the relative merits of Roentgen and radium therapy for benign conditions. In an attempt to determine which of these two methods is the most effective and what the actual results of treatment in practice are, they reviewed several hundred cases. They present an excellent discussion of pathologic considerations in which the mechanisms of aero-otitis and secretory otitis are described. The treatment of choice in hyperplasia of the lymphoid tissue is radiation therapy and the radium applicator is easily applied to the nasopharynx with the aid of local anesthesia. Beta Roentgens extend only about 1 cm. and are thusly absorbed.

Garland and associates believe that the simplest and safest method of irradiation of lymphoid tissue in the nasopharynx is external administration of Roentgen rays. The only disadvantage of this type of therapy is that six treatments are necessary instead of the usual three with radium. Anesthetization is not required, and children readily submit as soon as they learn that the initial treatment is painless. The great advantage of Roentgen therapy is that the whole lymphoid bearing area of the nasopharynx is included in each treatment. Usually six treatments are given at weekly intervals. The technique is described. Garland and associates review the results of radium treatment reported in the literature and present their results of Roentgen therapy in 720 cases of lymphoid hyperplasia. Fifty-five per cent of adults with deafness secondary to hyperplastic lymphoid tissue were cured with a single course of treatment and 60 to 90 per cent were relieved of presenting signs or symptoms.

If Roentgen therapy is as good as proclaimed by these authors, radium therapy would be quickly discarded, and all patients with lymphoid hyperplasia together with their complications would be quickly cured. This is a most excellent and rather convincing article; however, the impression is obtained that the authors are somewhat prejudiced.

Hardy and Bordley[22] give a report on a screening program in the public school system of Baltimore in which the use of the nasopharyngeal radium applicator in cases of lymphoid hyperplasia of the nasopharynx in children was tested. This report consists entirely of testing methods used, methods of analysis of results, and the like, and does not give any clinical data or results.

Ballantyne[23] studied 48 cases of deafness due to Eustachian tube obstruction encountered over a period of 18 months. In 37 cases one tube was irradiated in the usual manner and the other ear maintained as a control. Eleven patients had no treatment. Audiograms were obtained one month, three months and six months later. The results were contrary to what one would expect. In the 37 cases in which one ear was

treated, improvement occurred as often in the untreated as in the treated ear. Improvement was noted in 60 per cent of the treated cases and in 82 per cent of the untreated cases.

Weisman[24] attributes the occasional failure of tonsillectomy and adenoidectomy to produce the desired results of well-being to the fact that even after an expertly performed operation, remnants of infected and hypertrophied lymphoid issue remain. He describes Waldeyer's ring and adds that frequently lymphoid tissue is seen as discrete follicles on the posterior pharyngeal wall. The nasopharyngeal Beta radium applicator has been used in the treatment of some of these cases. The small localized area affected by the radium applicator limits its usefulness in many cases. Because of the ease of application, Weisman prefers Roentgen therapy directed to the nasopharyngeal region, and since lymphoid tissue is extremely radiosensitive, this tissue melts away long before any noticeable effects are produced on any of the neighboring structures.

SURGERY.

Reeves[25] points out that hypertrophic salpingopalatine folds, or lateral pharyngeal bands, may cause nasal obstruction even after an adequate adenoidectomy. Two cases are presented in which relief was obtained by removal of these folds with an adenoid punch.

Conway[26] gives a brief historical review of procedures used to try to attain normal speech in cases of cleft palate. He reports his experience in the handling of 38 complicated cases, in all of which at least four previous operations had been performed. He considers the use of a combined "push back" procedure and an inferiorly attached pharyngeal flap to be the most successful technique. In this procedure the pharyngeal flap is permanently fixed to the soft palate.

Hitschler[27] presents a case of a broken needle in the tonsillar fossa. Attempts to recover the needle at the time of the operation and some time later by biplane fluoroscopy were

unsuccessful. Four years later no symptoms had developed. Methods of prevention of such an accident, means of recovery of needles and possible complications are discussed.

Since good results have been reported with a dilute solution of procaine given intravenously for a variety of traumatic and painful conditions, Somers[28] decided to test its efficacy in the relief of post-tonsillectomy pain. Procaine in 1 per cent and 2 per cent concentrations was administered in different amounts to 41 tonsillectomized soldiers. The morning following operation, although some analgesia was obtained, and there were no severe reactions or deaths, the toxic effects were severe enough to preclude this type of analgesia in post-tonsillectomized patients.

Pearlman and Loewy[29] analyzed 45 cases of secondary hemorrhage from the tonsils and adenoids in 1,035 cases of tonsillectomy and adenoidectomy as to the barometric pressure changes at the time of hemorrhage. No correlation was established, but on the basis of their observations they state that "it is conceivable that the time of occurrence may be altered by prevailing conditions of these factors."

The nasopharynx is one of the most inaccessible areas of the body for diagnosis and treatment. Various surgical approaches to this area have been suggested, but most of them have been unsatisfactory. Wilson[30] describes a new surgical approach which has proved satisfactory in his hands. A transverse incision is made in the palate just anterior to the posterior margin of the hard palate and is extended downward at either end along the pterygomandibular raphe. The posterior spine of the hard palate is fractured, the tensor palatinus tendons are cut, and the whole soft palate is retracted posteriorly to expose to view the nasopharyngeal and posterior choanae. With this approach any surgical procedure desired can be performed in this area. The incision is readily sutured and there is no postoperative disability.

Sheridan[31] points out the difficulties of doing a complete adenoidectomy. He advocates the use of a bent metal suction

tip as a palate retractor and a 2 cm. laryngeal mirror to provide a better view of the nasopharynx, both before, during and after adenoidectomy.

BENIGN TUMORS.

Kerwin[32] reports a case of juvenile nasopharyngeal angiofibroma in a boy 16 years of age which was successfully treated with Roentgen-ray and androgen therapy. This is a rare type of tumor occurring in adolescent boys, which regresses as maturity is reached. The tumor is a benign fibroma, but because of its inaccessible position and sometimes rapid growth, it may be locally destructive. The main symptoms are hemorrhage and nasal obstruction. Because of the danger of serious hemorrhage, biopsy is not always advisable. Since spontaneous regression can be expected with maturity, the most conservative therapy should be employed. Kerwin considers radiation and hormone therapy as the best methods of control. Large tumors may be resected by electrocoagulation and recurrences may be controlled by interstitial irradiation and androgen therapy.

From a histopathologic aspect, nasopharyngeal fibromas are considered as benign tumors, but from an invasive point of view, Paredero del Bosque and Sebastian[33] are of the opinion that they behave as malignant growths. From a review of the literature they concluded that nasopharyngeal fibromas are extremely rare in England, not too uncommon in America and common in Egypt. These fibromas usually occur in males between 10 to 25 years of age. They commonly show spontaneous regression with oncoming maturity. The various methods of treatment are reviewed, and the conclusion is drawn that implantation of radium and repeated electrocoagulation yield the best results. Nasopharyngeal fibromas are radioresistant. Because of extensive and dangerous hemorrhage, surgical excision is seldom if ever used. Four cases encountered in the last three years are reported. This is a splendid article and valuable because of the review and compilation of opinions of outstanding men in this field.

Jepson[34] presents an unusual case of a persistent chordoma of the nasopharynx in which treatment over a long period of time, both by Roentgen-ray and irradiation and by surgical removal, resulted only in temporary benefit. Chordomas are tumors arising from embryonic remnants of the notochord. They occur at either end of the notochord and rarely are found in the nasopharynx.

Hoogland[35] reports an extremely rare case of nasopharyngeal cyst of branchiogenic origin in a man 69 years old who complained of pain and deafness in the right ear for six weeks. Examination disclosed a soft, fluctuating, cyst-like tumor the size of a chestnut in the right side of the nasopharynx pressing upon and obliterating a view of the Eustachian orifice. Aspiration of this fluctuating mass yielded 10 cc. of brownish opaque fluid. Ten months later the patient still had no complaints and re-examination yielded negative results. A diagnosis of nasopharyngeal cyst originating from the first branchial pouch was made. It was later determined that 45 years before the patient had a mass below the right auricle, which was considered at that time to be inflammation of the first external branchial cleft. This abscess subsided after incision and drainage. Guissani reported a similar case in 1928.

Hlavacek[36] reports an unsual case of a woman, aged 45 years, in whom a large hypopharyngeal polyp developed in the mouth after an attempt at strangulation. She had had no previous difficulty in swallowing, but the growth caused suffocation, which was relieved by its removal. The polyp was 21 cm. long with a peripheral circumference of 7 cm. It was club-shaped and attached posteriorly to the cricoid cartilage. Histologic examination revealed mature lipomatous tissue.

Penha[37] reports a case of nasopharyngeal fibroma in a hemophiliac patient with repeated hemorrhages which failed to respond to routine general and local treatment. He recalled that the natives of Portugal used the nettle urtica membranacea to stop bleeding, either by infusion or local application of the baked leaves; therefore, when all other measures failed,

Penha employed this common plant which promptly controlled the hemorrhage. This same hemostatic agent has been used successfully in other cases. Penha is conducting extensive investigation to determine the full therapeutic value of this plant, not only in cancer but also in leucemia.

Finerman[38] presents the case of a girl 13 years of age who complained of nasal obstruction of two years' duration, which was caused by a growth in the nasopharynx, proved by biopsy to be a juvenile nasopharyngeal fibroma. Considerable reduction in the size of the tumor was noticed six weeks following administration of 1,650 r of Roentgen-ray. Six months later the possibility of radon implantation was being considered, as surgical excision was being delayed as long as possible. This case is of interest not only because of the rarity of nasopharyngeal angiofibromas but also because some authors have challenged the occurrence of this type of growth in females.

MALIGNANT TUMORS.

In spite of our increased knowledge of nasopharyngeal carcinoma, Hickey[39] believes that the condition is too frequently overlooked. For this reason he presents a brief review of pertinent literature to indicate the trend of changing concepts of the disease and a summary of data from a survey of 25 cases in tabular form. The fact that only three have survived more than five years indicates that despite refinements in diagnosis and treatment "one frequently finds oneself baffled and somewhat helpless in achieving more than palliative control of the primary lesions and their metastases."

In an excellent and comprehensive article concerning Roentgen therapy of carcinoma of the hypopharynx, Jacobsson[40] presents an analysis of 322 cases treated beween 1939-1947, 75 per cent of which were in the lower part of the hypopharynx or below the level of the superior border of the cricoid cartilage. Sixty per cent of the series were females, in 90 per cent of whom the Plummer-Vinson syndrome was present. Treatment consists of 5,700 to 6,000 r over a period

of 28 to 30 days. This is given through four ports and is guided by beam direction through fluoroscopy. An overall five-year cure rate of 14 per cent was obtained. The five-year cure rate for lesions in the lower hypopharynx was 17 per cent. These results are much better than those obtained in other series.

Rojas[41] reports a case of lymphoepithelioma of the nasopharynx, which was first manifested by glandular enlargement on the right side of the neck. Further examination revealed a mass occupying the greater part of the nasopharynx and biopsy established the diagnosis. Roentgen therapy was immediately instituted. After 6,200 r were given in 29 days, the tumor and the cervical enlargement disappeared. Two years later adenopathy developed on the left side of the neck. Cortisone was given for 19 days, after which acute anemia developed, necessitating transfusions. More Roentgen therapy was given and all symptoms disappeared.

Whereas this is an interesting report, it is probably premature as far as cure is concerned; moreover, the value of cortisone must be questioned as in all probability the good results were accomplished by Roentgen therapy.

Epstein[42] advocates laminography in the diagnosis of nasopharyngeal tumors, and he points out that their clinical diagnosis is usually delayed until the appearance of symptoms referable to the eyes, ears, or upper nasal passages, such as diploplia, nasal discharge, nasal blockage, headaches, poor hearing and palpable lymph nodes. These types of tumors usually originate in the soft tissues of the nasopharynx and radiologic identification of alterations in these soft tissues is only occasionally mentioned as an important diagnostic sign. Epstein reports 10 cases, the youngest being a boy of 14 years with a nasopharyngeal fibroma and the oldest a woman 67 years old with a large lymphoepithelioma. Laminography is ideal for radiologic investigation of the nasopharynx. The technique is well described, and the reported cases demonstrate the existing pathologic alterations.

In an excellent thesis for the American Laryngological, Rhinological and Otological Society, Inc., Miller[43] presents the results of a statistical analysis of 102 cases of nasopharyngeal cancer encountered at several hospitals. Although his results compare favorably with those of other reported series, this paper is an excellent discussion of this subject and should be read in the original form by neurologists, ophthalmologists and general practitioners as well as otolaryngologists. Familiarity with the signs and symptoms might help to reduce the great time lag between onset of symptoms and diagnosis of nasopharyngeal cancer.

In addition to a rather complete historical review, a discussion of the histopathology, incidence, pathogenesis and symptomatology of the disease is included. The diagnosis can almost always be made by mirror nasopharyngoscopy with the use of the electric nasopharyngoscope. The commonest chief complaints are nasal obstruction, cervical adenopathy and unilateral deafness. Cervical metastases are present at the time of diagnosis in from 40 to 77 per cent of cases. Treatment is confined to the use of Roentgen-ray or radium, or both, and results are poor; however, the five-year cure rate has increased tremendously in the past 15 years, some authors reporting as high as a 25 per cent five-year cure rate.

Schoolman[44] urges careful routine examination of the nasopharynx in order to make an early diagnosis in cases of malignancy of the nasopharynx. Close cooperation between otolaryngologist and radiologist is essential in the treatment of these cases. Prognosis is better in the more radiosensitive tumors.

Stock[45] reports the results of a study of the effects of nitrogen mustard and urethane on nasopharyngeal cancer. Twenty-five patients with late stages of carcinoma of the pharynx were treated with 0.1 mg. of nitrogen mustard per kilogram of body weight daily for six doses. In a few cases there was extreme diminution in the size of the gland, and in advanced cases with bony involvement temporary decrease in pain;

however, in no case did this temporary remission last longer than four weeks. In most cases symptoms were not alleviated and the size of the tumor was not affected.

Twenty-three patients with hopeless nasopharyngeal cancer were treated with urethane. After an intravenous drip transfusion of 5 per cent glucose in water had been running for a few minutes, 10 gr. of urethane dissolved in 20 ml. of water were injected into the tube. The urethane was then washed through the vein quickly and the drip continued until 1,000 ml. of 5 per cent glucose had been given. The immediate results in these cases were as follows: five showed great improvement, four slight improvement, four no improvement, five refused adequate treatment and five died; however, Stock stated, "So far as can be seen at present, the effect is only temporary, for in most cases where some benefit has been shown, the tumor has recurred fairly rapidly."

Hynes[46] reminds us that extensive cancer involving both larynx and pharynx presents a difficult therapeutic problem for which there is as yet no satisfactory solution. Usually such patients are treated by radiation. Of 21 patients thus treated prior to 1945, only one survived. Twenty of these cases were candidates for extensive radical excision. Three patients not included in the radiation series had unilateral neck dissection and pharyngolaryngectomy, two of whom left the hospital in good condition, and the third was convalescing at the time of the report. The surgical technique, which is described in detail, represents a formidable procedure, but unless such methods are adopted these patients are doomed. The salvage is such that the procedure is warranted, and it is hoped that more laryngologists will follow Hynes' practice.

Martin and Quan[47] present statistical evidence of the racial susceptibility of the Chinese for nasopharyngeal cancer. They suggest as an explanation for this a racial or hereditary tendency rather than an environmental one, as suggested by some. From a survey of the literature and analysis of clinical and statistical data from the Memorial Hospital, they believe there is suggestive evidence that this susceptibility may be shared by all Orientals except the Japanese.

A rare and interesting case of spontaneous pneumocephalus in a patient with extensive carcinoma of the nasopharynx is reported by Raider.[48] The collection of air was apparently both cortical and ventricular, having entered the brain through a defect in the base of the skull caused by the malignant growth. The patient recovered from the pnenumocephalus without untoward effect; but, of course, the carcinoma was not cured, although the patient was alive and showed little change 14 months later.

Seltzer[49] gives a brief discussion of the various types of neoplasms of the nasopharynx. These include nasopharyngeal fibroma, squamous cell tumor, lymphoepithelioma and lymphosarcoma. The most suitable treatment for these lesions is irradiation.

In an interesting and comprehensive article, Digby[50] calls attention again to the fact that the pattern of nasopharyngeal carcinoma varies in different parts of the world. He thinks this is due to the differences in climate, environment, innate hereditary characteristics, customs and habits of the inhabitants. He cites the prevalence of nasopharyngeal carcinoma in Hong Kong, where it is the second commonest variety of malignant disease. In England and North America, nasopharyngeal carcinoma forms only a small fraction of 1 per cent of all cases of malignant disease, whereas in Hong Kong it constitutes 18 per cent of all malignant cases. Many theories have been advanced to explain this high percentage of nasopharyngeal malignancies in the Chinese, but none has been definitely proved. In addition to an excellent discussion of the pathology, Digby describes his method of establishing a clinical diagnosis, which is certainly unique. As in any type of carcinoma, the first principle of treatment is that early diagnosis is essential. The presence of any one sign referable to nasopharyngeal disease demands a thorough investigation of the nasopharynx. Radiotherapy is the best method of attacking these malignancies, since the cells are extremely radiosensitive and usually quick response is obtained. If the case is diagnosed early and treatment instituted immediately, good results may be expected. Unfortunately, too few cases

are seen early; consequently poor results are obtained in a high percentage of cases. Digby has presented his views and wide experience of years in Hong Kong in an interesting style. In view of the large number of cases he has seen, his opinions must be accepted as authoritative.

Thompson[51] reports four cases of lymphoepithelioma of the nasopharynx and discusses the symptomatology, diagnosis and treatment of this condition. A rather extensive discussion of the microscopic diagnosis of nasopharyngeal tumors is given. It becomes apparent from the divergent opinions quoted that the differential diagnosis of the various types of malignancy of the nasopharynx is extremely difficult. This is especially true in differentiating between lymphoepithelioma and transitional cell carcinoma; many pathologists consider them to be the same condition. This article should be read in its original form by those interested in this type of tumor.

Malignant neoplasms of the nasopharynx are largely epithelial in origin. Squamous cell carcinomas are the commonest, then adenocarcinomas and lymphoepitheliomas. Tumors of other origin consist of sarcomas, plasmocytomas and lymphocytomas. Extremely rare tumors in this area include chordomas, rhabdomyosarcomas and salivary gland tumors. In a clear, concise and well organized article, Omerod[52] discusses the signs and symptoms of these tumors, which he conveniently divides into four groups: nasopharyngeal, otologic, ophthalmoneurologic and cervical metastatic. A number of neurologic syndromes are described and typical case histories are presented.

Because of the rarity of radiation cancer in the deep tissue following external radiation, Goolden[51] reports four cases of patients in whom malignant tumors of the laryngopharynx developed following irradiation of the neck for benign conditions. In each case 25 years elapsed between the radiation therapy and the diagnosis of radiation cancer. A long latent period is characteristic of radiation cancer. In most cases of radiation cancer gross changes in the overlying skin are present.

Heck and McNaught[54] report two cases of juvenile fibroma which were successfully removed through a transpalatal approach. They point out that the advantage of such an approach is that postoperatively the structure is essentially normal as contrasted with the permanent disruption of nasal structures following the transantral approach.

Gynning[55] has treated five patients with carcinoma of the hypopharynx by the rotation Roentgen therapy method which is commonly used in carcinoma of the midesophagus. Two of these are free of symptoms over seven years after treatment. These two cases are reported and the technique, dosage and measurements of the method are discussed.

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ELECTROCOAGULATION OF THE VESTIBULE IN GUINEA PIGS.*†

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Vertigo, tinnitus and deafness are the most prominent symptoms of the condition known as "hydrops of the labyrinth." Conservative medical treatment may be directed toward improving the basic pathological condition or, when medical treatment is ineffective, the function of the affected ear may be eliminated by radical surgical treatment. This radical surgery is justified by clinical observations and experiments. Vertigo, one of the most disturbing symptoms, is due to an imbalance in the activity of the two labyrinths. If the activity of the abnormal ear is eliminated, the vertigo improves rapidly and ultimately a stable compensation is reached. The experimental work of Bechterew[1] proved that the compensation occurs in the vestibular nuclei. When tinnitus is a serious symptom total destruction of the affected cochlea is required.

Several surgical techniques have been devised to eliminate the abnormal activity that is the basis of the vertigo and tinnitus. The commonest procedures are: *1.* sectioning the VIIIth nerve as in Dandy's[2] operation, *2.* electrocoagulation of the vestibule as in Day's[3] operation, *3.* removal of the membranous labyrinth as carried out by Cawthorne,[4] and *4.* alcoholic injection into the vestibule.[5]

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All of the procedures are usually successful in relieving both the vertigo and tinnitus, but following section of the VIIIth nerve, tinnitus may remain or else reappear several weeks after the operation. Sometimes after the other procedures the tinnitus may remain or the vertigo may be induced by particular movements of the head. This persisting vertigo is usually attributed to some sensory structure which has escaped destruction by the operation.

The original purpose of the present study was to standardize the procedure of electrocoagulation of the vestibule in such a way as to minimize the loss of hearing. Subsequently we realized that there is probably no advantage in preserving hearing in hydrops of the labyrinth because the disease has already caused such great hearing loss and in addition there is likely to be severe tinnitus unless all hearing is abolished. We undertook, nevertheless, to standardize the electrocoagulation in order to minimize the risk of unintentional injury of the facial nerve and, furthermore, we hoped through histological and pathological stduies to find the basis for the occasional persistence of vertigo and tinnitus following electrocoagulation of the vestibule.

METHOD.

Experimental Animals.

The experiments were performed on 57 adult guinea pigs. These animals were chosen because of their suitability for relatively rapid preparation for histological study and because our electrocoagulation methods had been standardized for guinea pigs.

Apparatus.

A Cameron Duo-Cauterodyne* provided with a special electronic switch for regulating the duration of current flow was used for the electrocoagulation. This cautery is a radiofrequency power generator operating at four megacycles. The electronic switch consists of a gas tube (No. 2051) with a

*This instrument was generously loaned by the Cameron Surgical Specialty Co.

RC timing circuit in its grid circuit and a relay in its plate circuit for turning on the B voltage supply of the Cameron Duo-Cauterodyne. The filament current was kept on continuously to avoid the delay of about two seconds required to warm up the filament when the power is turned on by the foot switch supplied with the instrument.

Two types of sounds were used for stimulation. For pure tones a General Radio Oscillator Type No. 1304-A was connected to an attenuator and the output was fed to a Langevin 101-C Power Amplifier, which was connected to a Permoflux PDR-10 Earphone. This earphone was connected by an aluminum coupler and a small length of plastic tubing about 3 or 4 mm. in diameter to a small speculum which was sewed into the guinea pig's external canal, forming a closed acoustic system.

The other system utilized all the equipment except the pure-tone oscillator. Instead of this, it employed a square-wave generator which feeds two sets of band-pass filters set to the appropriate frequency. Each set of filters has a nominal cut-off of 18 db per octave. The resulting wave form is a brief train of waves which rises to a maximum in three or four cycles and then falls back to a minimum value in three to 10 waves (depending upon the frequency). The repetition rate used for these "tone pips" was usually 20 pips per second.

Surgical Procedure.

The external semicircular canal was approached through a retroauricular incision. The facial nerve was exposed where it emerges from the Fallopian canal. Under a dissecting microscope the bulla was opened immediately above the nerve. An opening of about 5mm. in diameter gives a full view of the ampullae of the external and the superior semicircular canals. The external canal is easily identified by its close relation with the middle ear cavity and the facial nerve.

The otic capsule of the ampulla of the external semicircular canal was fenestrated. (In some cases the fenestra was made in the middle third of the external semicircular canal instead of at the ampulla. In these cases the approach to the middle

third was through a hole entering the bulla between the facial nerve and the styloid process.) The membranous labyrinth was then treated by one or another of the following procedures for surgical injury:

a. *Surgical injury.* A fine needle was introduced into the vestibule and swept from side to side in several directions in an effort to destroy all of the sensory structures. In a few cases the injury was deliberately confined to cutting the membranous labyrinth just at its junction with the crista and without damage to any sensory structures.

b. *Labyrinthectomy.* The membranous labyrinth was removed from the vestibule by means of a dental broach.

c. *Electrocoagulation.* The needle electrode was introduced into the labyrinth. (The shank of the needle was not insulated in any way.) In most cases the time of current flow was 0.5 second, but sometimes it was reduced to 0.2 or increased to 1.0 seconds.

The intensity of the current was always between 20 and 45 milliamperes.

In acute experiments cochlear potentials, both the cochlear microphonics and the action potentials, were observed before and after the surgical injury to the vestibule. The electroacoustic methods have been described elsewhere.[6] In chronic experiments the cochlear potentials were not observed at the time of operation, but only when the animal was sacrificed.

In the chronic experiments the operation was performed under aseptic conditions, and the animals were treated with penicillin for four days after surgery. In this group ether anesthesia was employed for the operation, but in all cases dial in urethane was used for anesthesia in the final tests.

Following the electrical studies the animals were perfused and fixed. The temporal bones were embedded in celloidin. Serial sections were cut and then stained with hematoxylin and eosin.

RESULTS: "ACUTE" EXPERIMENTS.

Twenty-six ears (20 animals) constitute the "acute" group in which the animals were sacrificed between a few minutes and 24 hours after the surgical injury of the labyrinth. The results of the various types of injury were as follows:

1. Surgical Injury.

Only two ears were injured with the needle without electrocoagulation. In one the membranous vestibule was destroyed; in the other the membranous labyrinth was cut close to the crista. Microscopic examination of the first ear showed that the crista of the external semicircular canal had been detached and the macula of the utricle was partially injured. No damage had been done to the saccule or to the crista of the superior or the posterior semicircular canal (see Fig. 1). The cochlea appeared entirely normal on histological exami-

Fig. 1. Injury by needle alone. Survival, three hours. (A) Vestibule, showing hemorrhage and some pieces of bone. Arrow points to the region from which the crista of the external semicircular canal had been totally detached. (B) Vestibule. (1) Normal crista of superior semicircular canal and normal macula of the utricle. Other sections show some injury of the utricle. Arrow, crista of the external semicircular canal projecting into the utricle. (C) Vestibule. Arrow points to the normal crista of the posterior semicircular canal.

nation. The cochlear potentials were recorded in this experiment before and after the fenestration. Tests were made only at the frequencies 500, 2,000 and 8,000 cycles per second. The greatest effect was produced at 500 c.p.s., where the threshold for both action potentials and cochlear microphonics was elevated by 10 and 16 db, respectively. At 2,000 and 8,000 c.p.s no change of as much as 15 db was produced. The "electrical audiograms" are shown in Fig. 2.

Microscopic examination of the second ear in which the external semicircular canal had been cut showed no damage

of the sensory structures either in the vestibule or in the cochlea. The electrical responses of the cochlea showed a mild loss of sensitivity at 500 c.p.s., very much like that in the previous ear.

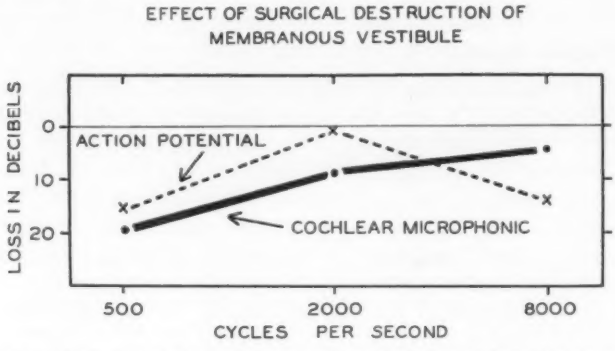

Fig. 2. The "thresholds" were determined before and immediately after surgical injury to the membranous vestibule.

2. Labyrinthectomy.

Four ears (three animals) were treated with this procedure. Microscopic examination confirmed the total removal of the walls and the crista of the external semicircular canal and of the walls and most of the macula of the utricle. In most cases the utricle was severely injured. The walls and crista of the superior semicircular canal were damaged or partially removed, but as a rule the crista of the posterior semicircular canal and the macula of the saccule were uninjured. The sensory structures of the cochlea were normal.

The effect of such labyrinthectomy upon the cochlear microphonic and on action potentials was a mild loss of sensitivity (30 to 35 db) at 500 c.p.s. and little or no effect at 2,000 and at 8,000 c.p.s.

3. Electrocoagulation.

A total of 20 ears (15 animals) were treated with electrocoagulation. In general this procedure caused necrosis of the crista of the external semicircular canal and the utricle.

Sometimes the crista of the superior semicircular canal was also involved. The extent of the necrosis depended upon the strength of the current. The injury produced by 20 milliamperes (for half a second) was mild, but 40 or 45 milliamperes caused severe injury. The mild damage was no greater than what can be produced by surgical injury without the help of the electric current. With the stronger current, however, the injury was definitely greater and presumably was caused by the current (see Figs. 3 and 4). Even with the stronger current, however, the crista of the posterior semicircular canal and the macula of the saccule usually escaped injury.

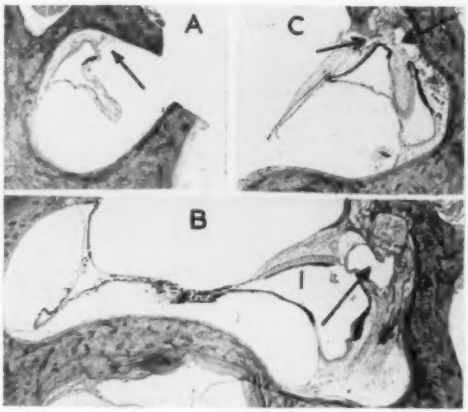

Fig. 3. Electrocoagulation by 40 milliamperes for 0.5 second. Survival, 0.3 hour. (A) Fenestra in external semicircular canal close to the crista. The latter is nearly detached and has been cauterized. Arrow points to the path of the electrode. (B, 1) Normal macula of the utricle. Arrow, hole made by the electrode. The macula has been partially detached and all of the tissue around the hole shows severe necrosis. (C) The arrows indicate the end of the pathway of the electrode. The macula of the utricle and the crista of the superior semicircular canal have been detached from the bony walls. Necrotic tissue surrounds the pathway.

The cochleas of this group of animals showed mild necrosis of some of the hair cells of the organ of Corti. The injury was most pronounced among the outer hair cells of the first and second turns. The stronger the current the greater was the extent and severity of the injury. In the experiments in

which the animal was sacrificed and fixed within the first 24 hours no structures in the cochlea other than the hair cells showed any signs of injury.

The cochlear microphonics and the action potentials after the operation usually showed a loss of sensitivity of 20 to 30 db at 500 c.p.s. when the coagulating current was 20 to 35 milliamperes. This current produced no significant effect on the thresholds for 2,000 and 8,000 c.p.s.

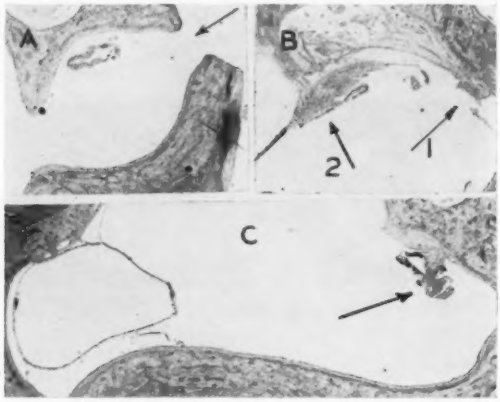

Fig. 4. Electrocoagulation by 45 milliamperes for 1.0 second. Survival, 1.5 hour. (A) Arrow points to fenestra in the external semicircular canal. The membranous canal has been reduced to a small necrotic mass. (B) Arrow 1 shows where the crista of the superior semicircular canal has been destroyed. Arrow 2 points to the partially destroyed and totally necrotic macula. (C) Arrow shows the totally necrotic crista of the exernal semicircular canal.

When coagulating currents of 40 to 45 milliamperes were used the loss of sensitivity for the electrical responses was slightly greater. Sometimes the responses at 2,000 c.p.s. were unaffected, but in other cases losses as great as 40 or 50 db were found both for the microphonics and for the action potentials. The responses at 8,000 c.p.s. were often practically normal. In other experiments, however, the threshold was raised by as much as 40 db for the microphonic and 80 db for the action potentials. Fig. 5 shows a case of such severe injury.

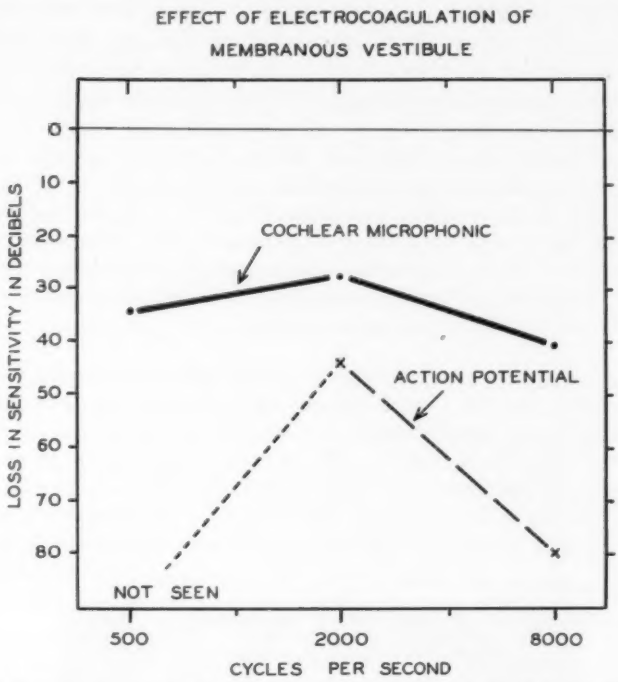

Fig. 5. The "thresholds" were determined before and again immediately after fenestration, followed by electrocoagulation of the vestibule with 45 milliamperes for 0.5 second.

RESULTS: CHRONIC EXPERIMENTS.

Twelve animals (15 experimental ears) were kept alive for periods from one to seven weeks after the operation. Considerable difficulty was encountered keeping these animals alive for the desired length of time. Twenty-five others died between the fourth and the eighth day after the operation, but the ears were not examined histologically because of the probability of autolytic artifacts. A few of the 25 animals died from pneumonia, otitis media, or meningitis. In others, the most probable cause of death seemed to be inanition, as the animals refused to eat and took very little water.

1. Surgical Injury.

Surgical injury was used in five of the animals in this group with a total of six ears. In three of these ears the fenestra was made in the ampulla of the external semicircular canal, in the other three it was made about the middle third of the canal. One of the ears fenestrated in the ampulla developed a suppurative otitis media and labyrinthitis. In this case all of the sensory structures of the cochlea and the vestibule were destroyed by the infection. The fenestra was closed by a mild fibroblastic and vascular proliferatnon coming from the periosteal layer. The fibrous tissue showed a pronounced infiltration by leucocytes.

In the second ear fenestrated in the ampulla the membranous canal was cut just at its junction with the crista but the vestibule was not injured. The animal was sacrificed eight days after the operation. Microscopic examination showed that all of the sensory structures of the vestibule and cochlea were normal. The fenestra (see Fig. 6) was closed by fibroblastic and vascular proliferation that had originated in the

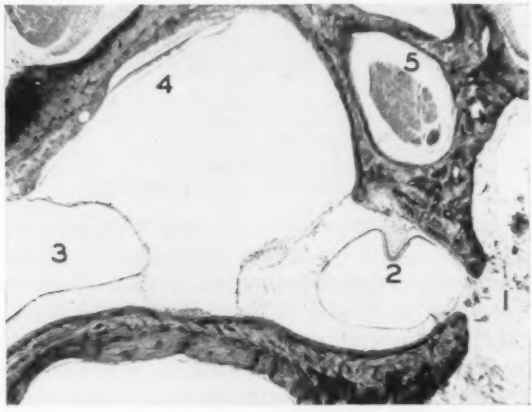

Fig. 6. Surgical section of the membranous canal close to the crista. Survival, eight days. (1) Fibroblastic and vascular proliferation blocking the fenestra. (2) Ampulla of the external semicircular canal, with normal crista. (3) Normal crus communis. (4) Normal saccule (the collapse of the walls is an artifact). (5) Facial nerve.

periosteal and endosteal layers. The enchondral layer apparently does not take an active part in the process of repair. Numerous bits of bone were found near the fenestra with signs of reabsorption and fibroblastic proliferation. Apparently the proliferation around some of the pieces of bone is due to the presence of either periosteal or endosteal tissue. Where the fibrous tissue is dense there are some signs of calcification. The healing process inside the ampulla presents superimposed and concentric layers of fibrous tissue which extend further along the canal than across the ampulla. The cochlear responses, both microphonics and action potentials, for this ear were within normal limits.

In the third ear that was fenestrated at the ampulla the vestibule was injured. The animal was sacrificed eight days after the operation. Microscopic examination later showed both an otitis media and the usual surgical damage to the crista of the external semicircular canal and the macula of the utricle, but the cristas of the superior and posterior canals and the saccule were not affected. The healing process at the fenestra was similar to that described in the previous case. All of the sensory structures of the cochlea were normal, and there was no sign of infection of the inner ear; nevertheless, the thresholds for both the cochlear microphonics and for the action potentials were elevated by about 55 db. The probable explanation of this hearing loss is the otitis media that filled the cavity of the inner ear.

In three ears fenestrated in the middle third of the external semicircular canal the membranous canal was destroyed with a fine needle that was introduced in the direction of the ampulla. The animals were sacrificed between three and four weeks after the operation. Microscopic examination showed that all of the sensory structures of the cochlea and the vestibule remained normal. The fenestra was blocked by fibrous and vascular tissue which filled the canal for a distance of about 1 mm. (see Fig. 7). The fibrous tissue extended just to the opening of the ampulla. Numerous bits of bone were present at the fenestra and in the canal. They showed signs of

resorption and fibroblastic proliferation, and calcification or formation of new bone could be seen in the dense fibrous tissue at the surface of the canal.

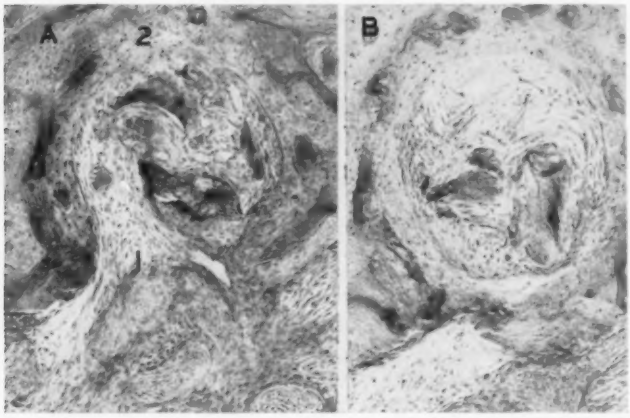

Fig. 7. Surgical injury by needle passed along the external semicircular canal toward the ampulla from a fenestra in the middle third of the canal. Survival, 20 days. (A, 1) Fenestra filled with fibrous tissue formed from the periosteum. The lumen of the canal is also completely filled with fibrous tissue, pieces of bone, and new bone. (2) Proliferation from the endosteum. (B) Same canal, about 0.75 mm. from (A). The canal is still filled by fibrous tissue and new bone, but the tissue is not so dense nor is the formation of new bone so active.

2. Labyrinthectomy.

No animals survived this procedure long enough to be included in the chronic group.

3. Electrocoagulation.

Seven animals (nine operated ears) constitute this group. In seven ears the fenestration was in the middle third of the external semicircular canal; in the other two the vestibule was coagulated through a fenestra in the ampulla. In all cases the current was 35 milliamperes.

The first animals were sacrifiecd at one, four and seven weeks after the operation. The three ears taken after one week's recovery showed no damage to the sensory structures of the inner ear and the healing process at the fenestra fol-

lowed the same pattern as already described. The electrical responses were not studied in these animals. The only ear taken at four weeks showed normal sensory structures in the vestibule and the usual fibroblastic proliferation and formation of new bone at the fenestra and in the semicircular canal. In this ear there was mild atrophy of the organ of Corti and Reissner's membrane was collapsed. The thresholds for both microphonics and action potentials were elevated about 20 to 25 db at 500, 2,000 and 8,000 c.p.s. One ear taken after seven weeks' recovery gave no electrical responses. Histological examination showed complete absence of hair cells throughout the organ of Corti. The supporting cells were normal in the apical turn but showed some atrophy in the first and third turns. In the second turn the organ of Corti was replaced by flat epithelium. The ganglion cells and the mesothelial cells were uninjured. Likewise the saccule and the crista of the posterior canal were normal. In this specimen, unfortunately, the section showing the utricle and the superior semicircular canal were lost. The other two ears taken after seven weeks' recovery showed no damage of the sensory structures either in the vestibule or the cochlea. The healing process in the fenestra (at the middle third of the semicircular canal) followed the same pattern as in the other chronic cases. The cochlear responses of these two ears were within normal limits.

The animal in which bilateral electrocoagulation of the vestibule was carried out through a fenestra in the ampulla of the external semicircular canal was sacrifiecd 10 days after the operation. It had developed bilateral otitis media and labyrinthitis on both sides. The sensory structures of both vestibule and cochlea had disappeared, but on one side the supporting cells of the organ of Corti still remained normal. These ears gave no electrical responses. These observations illustrate the special sensitivity of the hair cells to injury, in this case by infection, and also the well established dependence of the electrical responses on the integrity of the hair cells.

DISCUSSION.

Schall and Rambo[7] used a portable Bovie electrosurgical unit for coagulation of eight membranous labyrinths in five

monkeys. In one series of experiments the dial setting of the instrument was 20. The current was applied for one second and repeated within 10 seconds. In another series the dial setting varied from 10 to 40. These animals were sacrificed after 30 days, while the other series were allowed to survive for periods of 10, 20, 30 and 40 days. For a dial setting of 20 or more, they found the membranous labyrinth to be completely destroyed. With a setting of 10 the saccule and utricle were incompletely destroyed and the semicircular canals undamaged. They were unable to correlate damage to the cochlea with either length of postoperative life or intensity of the coagulating current.

In our own experiments the microscopic examinations of the temporal bones show that we did not succeed in producing complete destruction of the vestibule either by surgical injury or electrocoagulation. The crista of the posterior semicircular canal and the macula of the saccule remained even when coagulating currents of 40 to 45 milliamperes were used. Unfortunately we did not keep enough animals alive long enough to be sure whether these structures would survive permanently or whether they might have degenerated several weeks after the operation.

We have no reliable information as to strength of the coagulating current actually employed by various surgeons in Day's operation on the human. The results are sometimes unsatisfactory, because after the operation patients sometimes complain of a vertigo, most frequently brought about by nodding the head. This relation suggests that the posterior or the superior semicircular canal is still active. It is possible that one or both of these canals escape coagulation just as the posterior (and sometimes the superior) canal did in our guinea pigs. We believe that electrocautery should be applied several times to the vestibule and with various positions of the electrode in order to reach the posterior semicircular canal of the saccule. The intensity of the current must be carefully regulated because it has little effect below 44 milliamperes. An electrode insulated except for the tip should localize the current most effectively. The insulation would prevent con-

tact with bone at the fenestra and thereby reduce the probability of spread over the surface of the bone to the facial nerve.

In general the heating effect of a brief electrical current is related to its duration as well as to its intensity. If brief currents are to be employed, the duration must, therefore, be regulated accurately. The delay of two seconds required by the original Cameron Duo-Cauterodyne to warm up its filament makes it unsuitable for such brief dosage. The current does not rise to effective strength for two seconds after operation of the foot switch. We suspect that in many human operations in which similarly designed instruments are employed, no actual electrocoagulation is produced. The injury may be only from the insertion of the needle.

Our microscopic examinations show that a rapid fibroblastic proliferation occurs which sometimes fills the entire closed section of the semicircular canal after two to three weeks. This overgrowth originates from the periosteum and the endosteum. The great activity of the endosteal layer is probably stimulated by the surgical injury from the electrode. There is no such fibroblastic proliferation in the vestibule where the space is broad, and hence the injury to the periosteum is minimized.

Coagulating currents of high intensity elevated the thresholds of the cochlear microphonics and the action potentials. The elevation of thresholds occurred in the cases that showed necrosis of the hair cells, but the response to 500 c.p.s. was constantly affected whatever procedure was used and regardless of any abnormality of the hair cells. This effect is probably produced by alteration of the acoustics of the inner ear when the new fenestra is opened. The new fenestra will obviously act like an acoustic leak, and it is reasonable that its effect should be greatest at the lowest frequency.

SUMMARY.

Electrocoagulation of the vestibule, following the general procedure of the Day operation for Ménière's disease, was performed on 57 guinea pigs. A special electronic switch was

provided to limit the electrocoagulation to a predetermined time, usually 0.5 second. Electrical responses to sound were studied immediately or after days or weeks of survival. The temporal bones were later sectioned and examined microscopically.

Currents less than 40 milliamperes flowing for 0.5 second produced little or no more damage than the mere insertion of the electrode. Currents of 40 to 45 milliamperes produced necrosis of tissue near the needle track, but did not injure the posterior semicircular canal or the macula of the saccule. Sometimes partial injury of the organ of Corti was produced by such doses.

The elevations of electrical "thresholds," both cochlear microphonic and action potential, for frequencies 500, 2,000 and 8,000 c.p.s., correlate well with the degree of injury to the hair cells in various parts of the cochlea. The cochlea was sometimes intact and electrical responses normal four to seven weeks after surgical injury, with or without electrocoagulation of the vestibule with 45 milliamperes for 0.5 second.

The surgical fenestras in the labyrinth were closed by fibroblastic proliferation that filled the entire canal. New bone was present four weeks after operation. The endosteum as well as the periosteum seems to initiate this fibrosis and osteogenesis.

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LATE CONGENITAL SYPHILIS OF THE EAR.*†

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There are relatively few diseases of the labyrinth whose pathology is well understood that cause progressive profound hearing loss in the apparently healthy young adult. Malignant otosclerosis is one disease of this group; late congenital syphilis another. The detailed description of the clinical symptoms and the temporal bone findings in late congenital syphilis by Mayer and Fraser,[1] in 1938, laid a firm foundation for this disease entity and separated it from other syphilitic disease of the ear.[2] The main features in this disease are bone lesions of gummatous osteomyelitis. These may invade the labyrinth to produce serous labyrinthitis associated with miliary gumma in the endosteum and dilatation of the cochlear duct and finally pronounced degeneration of the cochlear and vestibular end-organs.

There may be productive inflammatory change in the perilymphatic space of the semicircular canals, sparing the endolymphatic space, and erosions of the endosteal layer of the bony canal may also occur. The ossicles and the annular ligament may be invaded as well as the modiolus. The histological appearance of the subendosteal periostitis of the semicircular canal indicated to Mayer and Fraser alternating periods of exacerbation and quiescence. While the incidence of this particular disease as a cause of deafness in the general population is now very low, the incidence in young adults with profound bilateral progressive hearing loss, excluding malignant otosclerosis, is significant. A re-examination of

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some of the problems associated with the diagnosis and treatment of this disease is presented. Only a few studies have appeared in the literature in the past decade.

The ear symptoms usually develop rather abruptly long after other active manifestations of the congenital luetic infection have subsided.

Despite apparently intensive early treatment and inactivation of early sites of infection, the temporal bone may suddenly become involved in a rapidly progressive disease during the second and third decade of life, even though the patient may be in apparently good general health. A clinician actively involved in the treatment of lues may recognize its various manifestations. The otologist, however, is usually consulted long after treatment has been terminated. Indeed, the patient may have little awareness of the nature of the early infection. Early manifestations may not be readily apparent. Deformed teeth (Hutchinson's teeth) may have been capped or extracted. Interstitial keratitis may have been very mild and after healing may have left very little signs. A careful examination with a slit lamp may be necessary to find evidence of the corneal scars. Blood serology may be equivocal or negative. Laird,[3] in analyzing 115 cases of late congenital syphilis in England, points out that the diagnosis may be obvious or extremely difficult and that one seldom sees infantile syphilis now. He found that over 80 per cent of the cases were diagnosed only after the first decade of life and usually because a disease appeared that suggested the need for a blood test.

While there were positive eye findings in 61 of his patients, bilateral nerve deafness was noted in only eight. Both sexes were equally affected in the eye disease, but seven of the eight cases of ear disease were in females.

The direct examination of the ear and results of the auditory and vestibular tests and X-ray were not diagnostic in themselves. In the presence of intact, relatively normal drums, the clinician would ordinarily not do a fistula test. The presence of a positive fistula response is a rather unique feature of this disease first described by Hennebert[4] in 1911. The

explanation for this phenomenon became clear later when the pathological changes in the temporal bone were described. A hastily done fistula test with only positive pressure may show no eye movement or the eye movement may be so brief as to escape notice. Typical nystagmus may be evident only on negative pressure. The nystagmus is usually of very short duration (one second) compared to that produced under similar pressure conditions in the ordinary labyrinth fistula (10 seconds). The gummatous invasion of the temporal bone marrow and blood vessel spaces is a striking feature of the disease as described by Mayer and Fraser. Both proliferating and destructive changes occur in the capsule bone. The labyrinth capsule bone may be riddled with miliary gumma even in the region of the annular ligament. Invasion of the ossicles themselves with secondary ankylosis of the incudomalleolar joint may add a conduction element to the deafness, but bony ankylosis at the stapes footplate was never seen. The inflammatory change extends to the labyrinth itself to produce various degrees of chronic labyrinthitis to account for the severe auditory and vestibular symptoms. The symptomatology, however, is not particularly distinct and in itself cannot suggest the diagnosis. Bilateral symmetrical profound progressive loss, often including low tones, without remissions and with various degrees of tinnitus and vertigo, is the clinical picture that these patients usually present. The findings in the patients studied are presented briefly, including audiograms and vestibular tests to help point out some of the problems in the diagnosis and treatment of this special luetic disease of the temporal bone.

Case 1: A. G., female, age 22, had onset of hearing trouble 11 years ago. This became handicapping within a few months. There was no tinnitus and only one episode of vertigo. Severe interstitial keratitis occurred at age six, requiring an iridectomy and producing some visual impairment. She received intensive antiluetic treatment at that time. Hutchinson's teeth were present. The blood and spinal fluid serology were negative while under observation in the clinic.

The audiogram demonstrated a profound nerve type deafness bilateraliy. There was a marked impairment of the vestibular response to the cold caloric test. The fistula test was positive to negative pressure in both ears on repeated tests over the period of five years during which the patient was under observation.

A course of fever therapy had no effect upon the hearing. The audiogram appeared to stabilize at a very high threshold (see Fig. 1). A hearing aid was very effective and relieved tinnitus. She was able to attend high school, get a job, and married.

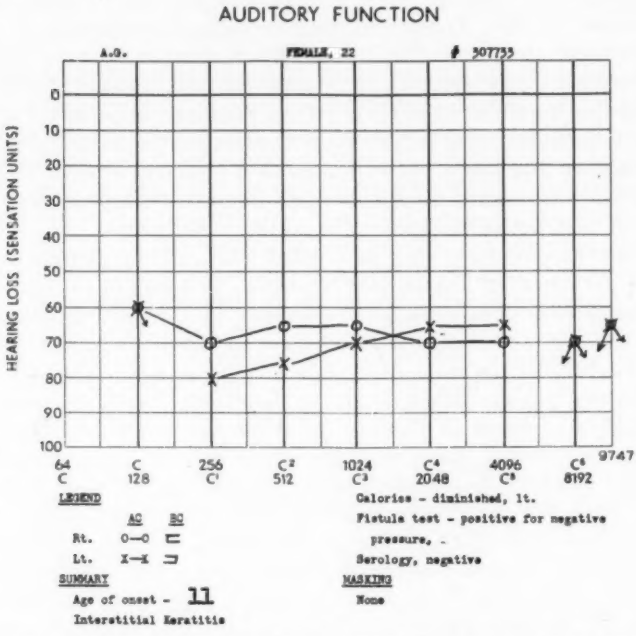

Fig. 1. Audiogram of young woman 11 years after the onset of ear symptoms in late congenital syphilis. The hearing continued to deteriorate in the face of antiluetic and fever therapy. No hearing by bone conduction could be elicited with the audiometer.

Comment: The response of the fistula to negative pressure is much stronger than that to positive pressure.

Based on their pathological findings, Mayer and Fraser explain the fistula response in three different ways:

1. A gummatous softening of the capsule from the outside and adjacent to the antrum.

2. A gummatous penetration of the bony canal from the inside leading into a narrow space.

3. A gummatous infiltration of the stapedial ligament producing abnormal mobility.

The production of eye movements on pneumatic movement of the intact drum and conducting mechanism is probably the result of unusual, abnormal mobility of the stapes due to invasion by granuloma of the capsule bone in relation to the annular ligament together with limited softening of the bony canal wall from the endosteal surface. This explanation is supported by the fact that negative pressure is more effective than positive pressure in producing nystagmus. Even the normal conducting mechanism is more easily moved by negative pressure than by positive pressure.

The short duration of the fistula response is also a noteworthy feature. Only two to three large movements lasting about one second can usually be induced even with maintained negative pressure. This suggests a different mechanism of vestibular irritation than that seen in the fistulas of the horizontal canal, where positive pressure is very effective in producing nystagmus and where the nystagmus lasts about 10 seconds with about 15 eye movements of gradually diminishing strength. Despite the severe cochlear involvement, the vestibular apparatus was not destroyed. The combined impairment of vision and hearing in this patient resulted in severe disorientation with her environment and led to an accident that killed her and her child. Unlike some of the patients in this group, this patient went through a pregnancy without change in her hearing, but cochlear function was profoundly affected.

Case 2: C. B., female, age 23, had onset of progressive deafness at age 17. Within three years, the hearing loss was severe and the patient began to have tinnitus bilaterally and dizzy spells. The hearing loss increased with these symptoms. Evidence of inactive interstitial keratitis was present. The serology has been weakly positive. Interstitial keratitis was actively treated at age of seven.

The audiogram shows a severe bilateral, rather flat perceptive hearing loss, while the minimal cold caloric tests were normal (see Fig. 2). The fistula test was equivocal.

The patient wears a hearing aid and received symptomatic treatment. Regular antiluetic therapy has been given repeatedly.

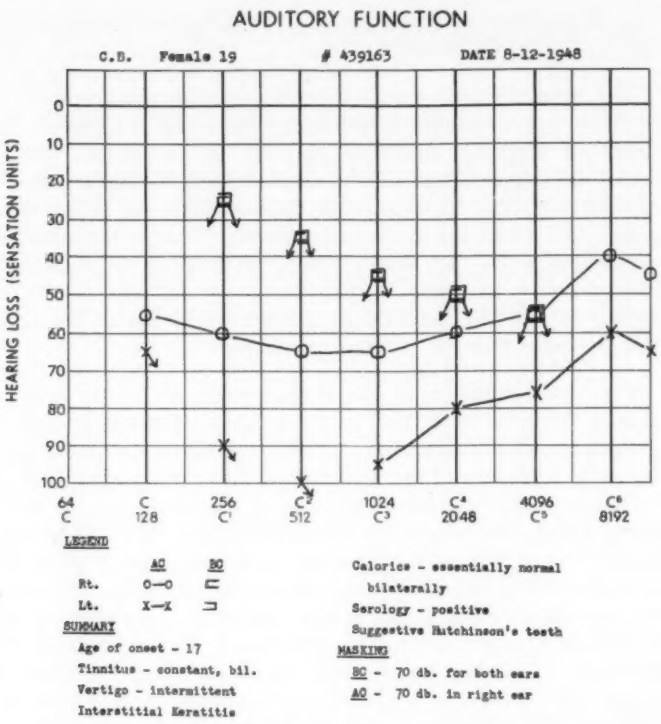

Fig. 2. Audiogram two years after onset of symptoms in a young woman with late congenital syphilis of the ear. Despite intensive antiluetic therapy, the hearing continued to deteriorate and two years later the cochlear function on the left was completely destroyed.

Comment: Repeated and thorough antiluetic regimes did not stop the progression of the cochlear involvement, and the hearing in one ear was completely destroyed.

Despite a normal caloric test, some disturbances in vestibular function were present in the patient, as indicated by the occurrence of attacks of vertigo.

Case 3: B. F., female, age 42, noticed a slowly progressive bilateral loss of hearing and slight tinnitus at age 18. At age 35, immediately following her first pregnancy, the deafness became profound bilaterally and the tinnitus increased. No vertigo occurred. Before the onset of ear

symptoms, the patient had bilateral interstitial keratitis which became quiescent after treatment. A positive blood serology and negative spinal fluid were found at age 18, while during the period of rapidly progressing deafness the serology was only weakly positive or negative on repeated tests.

The original audiogram done in 1945 showed a severe deafness bilaterally. Repeated hearing tests (see Fig. 3) showed a moderate advancement of the cochlear disease. The cold caloric test was reduced on the first examination and was normal on the second. On the right ear, a fistula response could be obtained on both positive and negative pressure.

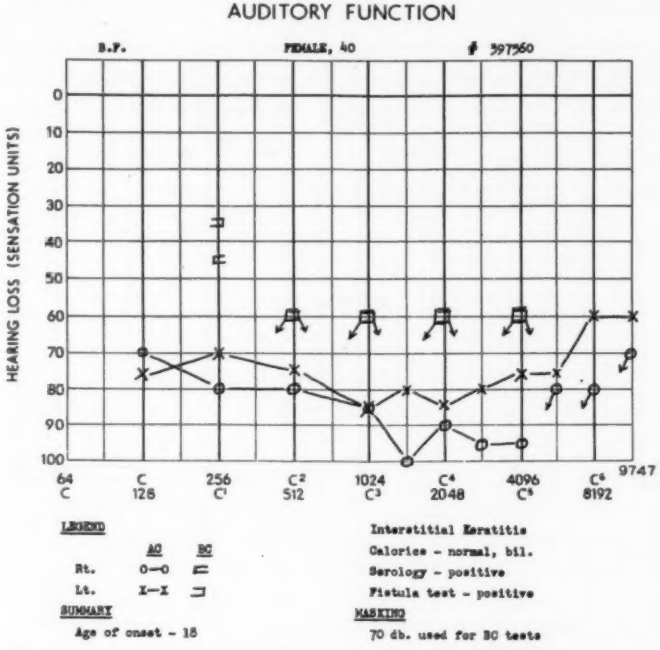

Fig. 3. Audiogram of patient with late congenital syphilis of the ear who noted a hearing loss for years, which rapidly became worse after pregnancy, and did not improve with intensive antiluetic therapy.

Active antiluetic therapy including bismuth, arsenic and penicillin during this time did not prevent continued deterioration of her hearing. She obtained help from a hearing aid.

Comment: The sudden severe exacerbation of her deafness shortly after pregnancy is noteworthy.

Case 4: R. M. L., female, age 22, noticed tinnitus and associated loss of hearing at age 17. The hearing loss increased, especially in the right ear, as the tinnitus subsided. The hearing loss was accentuated during and after a pregnancy. There was intermittent, transitory dizzy spells in the early period of the labyrinthine disease. Hutchinson's teeth were observed. The patient had inactive but handicapping luetic ocular disease that began at five years of age. The serology was negative after treatment at age 11. The parents' serology was positive.

A profound nerve type deafness was present bilaterally with a somewhat diminished response to the cold caloric tests. There was spontaneous nystagmus to the left on head shaking. The fistula test was positive to negative pressure on the left side.

Despite further antiluetic therapy, the hearing continued to deteriorate (see Fig. 4).

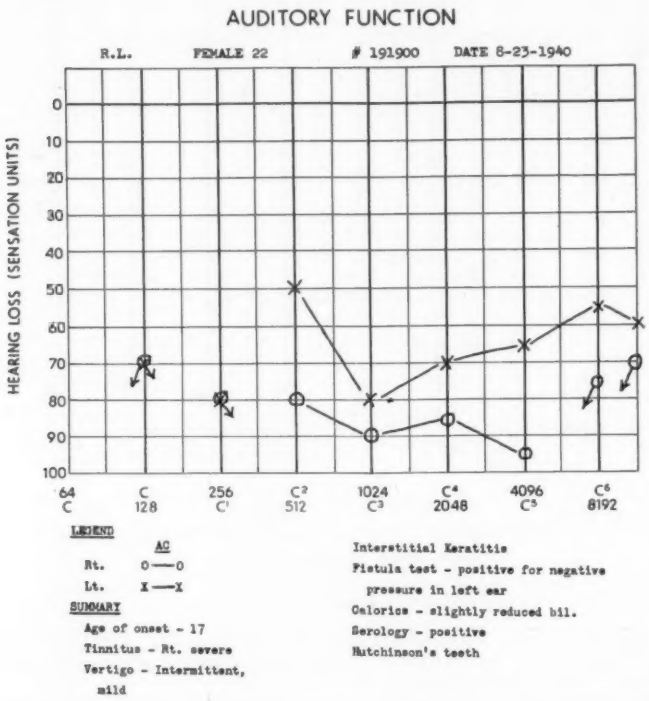

Fig. 4. Audiogram of young woman with late congenital syphilis of the ear five years after the onset of ear symptoms. Hearing by bone conduction was profoundly affected. Antiluetic therapy did not stop the progression of the ear disease.

Comment: The progression of deafness in face of intensive antiluetic therapy is commonly observed. Tamari[5] reported that despite the administration of 9,000,000 units of penicillin in 15 days, both the hearing and the vestibular function continued to deteriorate.

On the other hand, when the labyrinth function is profoundly affected, reversal of the labyrinthine pathology by any treatment is not likely.

The profound cochlear loss in this patient contrasted to the mild vestibular impairment is in line with the clinical observation of apparent greater sensitivity of the auditory end-organs over the vestibular in labyrinthine inflammations. The active fistula response to only negative pressure and the history of pregnancy influencing the progression of the deafness are again noteworthy.

Case 5: C. T., female, age 40, noted tinnitus and loss of hearing in both ears at age 35. Following pregnancy at age 38, the hearing loss and tinnitus increased markedly. Dizzy spells and headaches followed. Suicide was contemplated. The serology has been weakly positive. Interstitial keratitis occurred in the late teens but cleared quite well.

Cold caloric tests showed reduced sensitivity of the right vestibular apparatus only. The fistula response was present on negative pressure in the right ear. Intensive antiluetic therapy did not prevent deterioration of hearing.

In July of 1951, the patient received 100 mgm. of ACTH per day for seven days while on 600,000 units of penicillin daily for 10 days. Pure tone acuity and speech discrimination thresholds were greatly improved.

This improvement has been maintained (see Fig. 5). With the improved hearing, there was an associated decrease in dizzy spells and headaches.

Comment: Again pregnancy is seen to influence the activity of the disease. Therapeutic problems are discussed below.

Case 6: V. A., female, age 26, was well until the age of 23 when there was onset of severe tinnitus and frontal headaches. These persisted for several months before decreased auditory acuity was notice on the left side, followed by involvement of the right ear in a few months. The hearing loss became progressively more severe in the months that followed. The patient then began to have intermittent dizzy spells and noted an impaired sense of balance, more marked at night or in a dimly illuminated room. There was a history of keratitis at age of four. The patient's mother had received antiluetic therapy just prior to the patient's birth.

General examination revealed a widespread vitiligo and a white patch of hair at the nuccal line. There was a large inactive central perforation in the left eardrum.

Except for inactive interstitial keratitis, iridocyclitis, vitiligo and poliosis, the general examination was negative.

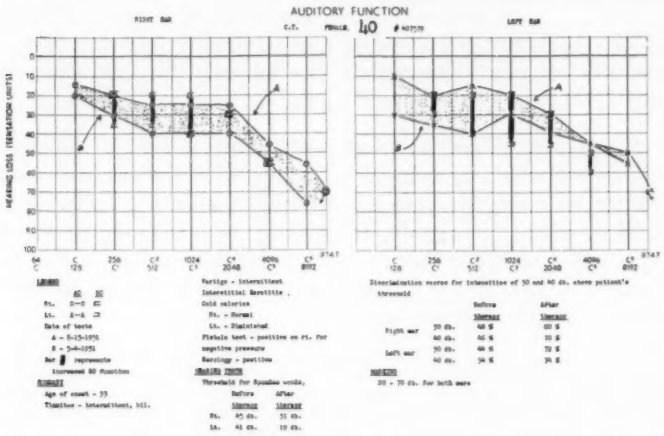

Fig. 5. Audiograms of a patient with late congenital syphilis of the ear with five years of progressive symptoms, markedly increased after pregnancy three years later and showing maintained improvement of function after a course of combined penicillin and ACTH therapy.

There was a bilateral nerve type deafness, more severe on the left side, and bilaterally positive Rinnes with the Weber referred to the right ear. There was no caloric response to ice water on the right and none to cold alcohol on the left. There was a spontaneous nystagmus to the right. She had considerable difficulty in walking with the eyes closed. The fistula test was negative at this time.

The blood serology was positive, while the spinal fluid was negative. In view of the poliosis, vitiligo, uveitis and deafness, a diagnosis of congenital lues and possibly Vogt-koyanagi's syndrome was made.

Unlike the other patients in this group, this patient, after a full course of treatment with bismuth and arsenicals, showed an improvement in the right or better hearing ear. A positive fistula reaction was found at this time on the right side, both to positive and negative pressure. In March, 1952, she received a combined course of ACTH and penicillin because the hearing had again deteriorated. Twenty days later the threshold of the right ear was superior to any obtained in previous tests (see Fig. 6). Tinnitus first decreased and then fluctuated a few days after therapy was started.

Comment: Uveoparotid fever, Cogan's and Vogt-koyanagi's syndromes may be associated with inner ear pathology pro-

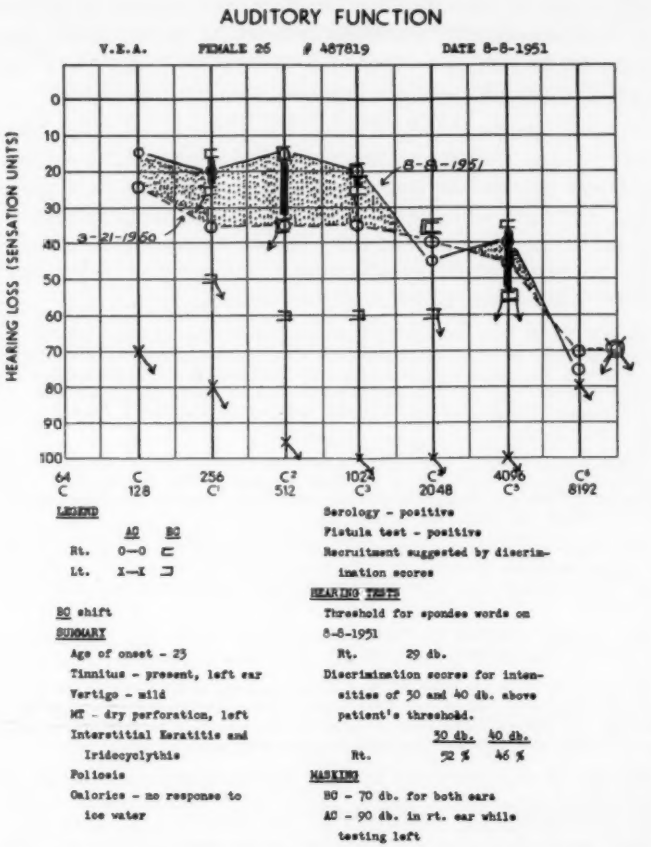

Fig. 6. Audiograms of a young woman with congenital syphilis showing total loss of cochlear function on the left side and moderate loss on the right after about three years of symptoms. The improved function in the right ear after a combined course of penicillin and ACTH therapy is represented. Some improvement was obtained before this by a course of bismuth and arsenicals.

ducing hearing and vestibular loss resembling that seen in late congenital syphilis and were seriously considered in the differential diagnosis in this case; however, the eye and ear symptoms in the above diseases are usually concomitant or

follow one another rapidly. Poliosis and vitiligo is part of the Vogt-koyanagi's syndrome and would be the most likely diagnosis in this case except for the typical history and findings of congenital syphilis and the positive fistula test.

In considering the differential diagnosis in all these cases, one may also note that some familial diseases of the ear may manifest themselves long after birth to produce in a rather short time marked auditory and vestibular symptoms in the second to fourth decade of life. These are inherited degenerative processes probably affecting the inner ear, often associated with other systemic disturbances.

Case 7: M. B., female, age 21, had onset of tinnitus at age nine. Within two years, the hearing acuity in both ears had decreased to a handicapping level. The tinnitus persisted, and in the late teens, transitory vertigo occurred. The patient could make herself dizzy by pushing on the right tragus. Bitemporal and parietal headaches have been severe in the past three years.

There was a history of a positive serology in the patient at the age of nine. Her mother had had a positive serology. The patient had interstitial keratitis, but this is now inactive. The patient's blood and spinal fluid serology are negative at present.

Hearing tests showed a profound bilateral perceptive deafness (see Fig. 7). The response to the cold caloric test was reduced only in the left ear. There was a slightly positive fistula test to negative pressure in the left ear.

The patient received a combined course of ACTH and penicillin for seven days with no effect on auditory symptoms or function.

Case 8: A. C., male, age 24, had onset of deafness in the right ear at age 12. This progressed in a few years to an inadequate level and soon the left ear became involved. A hearing aid was fairly satisfactory until age 23 when the hearing suddenly deteriorated with an acute "flu."

The patient had interstitial keratitis in 1938, several years before the beginning of ear symptoms, and received antiluetic treatment at that time. The mother was a known luetic.

The routine clinical examination was noncontributory. Hearing tests showed apparent total loss of cochlear function, yet tinnitus was present and sometimes was severe (see Fig. 8). There was no response to ice water in either ear. The fistula test was questionable in the right ear.

A course of continued ACTH and penicillin treatment was given without effect.

Comment: When the gummatous lesions reach the labyrinth spaces, a rapid progression of symptoms may be expected as labyrinthitis rapidly develops. The profound loss of vestibular function together with apparent total hearing

loss indicates a very extensive irreversible labyrinthine infection. The presence of severe tinnitus despite the absence of cochlear function is noteworthy.

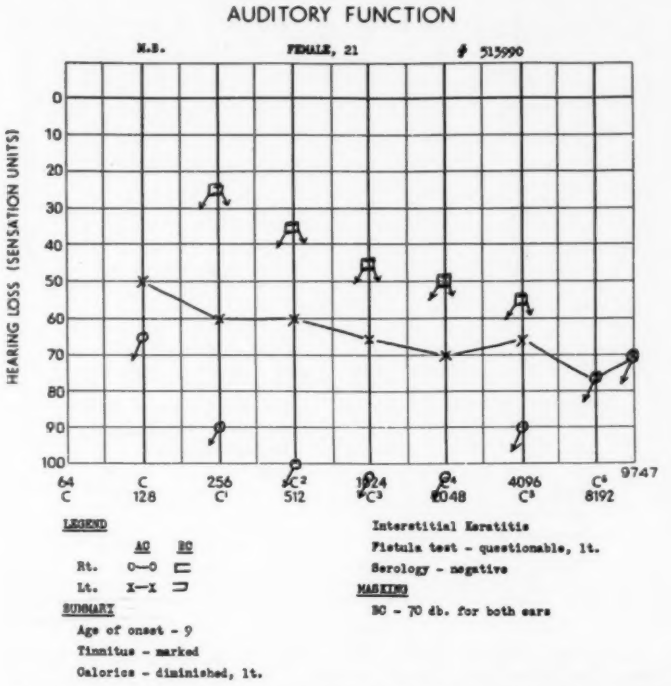

Fig. 7. Audiogram of a young woman with 12 years of progressive ear disease due to congenital syphilis not changed by combined penicillin and ACTH therapy.

Case 9: N. P., female, age 29, had onset of progressive deafness at 19 years of age. Initially the deafness involved the left ear but, following a pregnancy in 1947, the hearing in the right ear dropped. The bilateral nerve type deafness then remained essentially stationary. Tinnitus was relatively mild and history of vertigo was vague. Associated with the hearing loss was an inactive luetic interstitial keratitis. Positive blood serology and negative cerebrospinal fluid serology were found.

Inactive middle ear disease was camouflaged by the profound nerve type deafness. The fistula test was strongly positive on negative pressure on the left side and weakly positive to positive pressure.

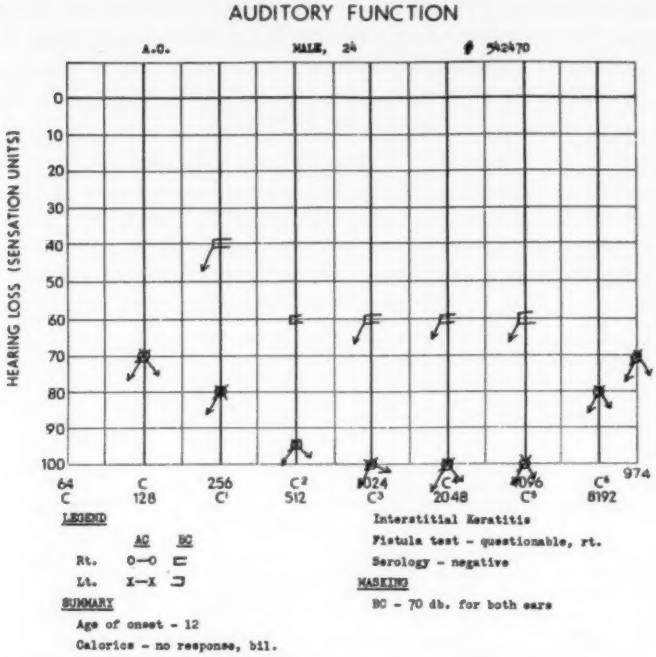

Fig. 8. Audiogram of young man with 12 years of progressive ear disease due to congenital syphilis leading to total destruction of cochlear function. No response was obtained by a course of combined penicillin and ACTH therapy.

Arsenic and bismuth therapy was ineffective in changing the hearing acuity. The patient was treated recently with ACTH and penicillin. The pure tone threshold curves were not changed, but speech discrimination tests suggested a significant improvement in the ear with the poorer threshold curve (see Fig. 9).

Case 10: E. H., female, age 41, had onset of vertigo with tinnitus and hearing loss at age 33 while receiving antiluetic therapy for a second attack of interstitial keratitis. The dizzy spells often seemed related to position changes. Nystagmus to left with vertigo lasting about two minutes when the patient is lying on the right was noted on Aug. 9, 1950. With each dizzy spell, the hearing seemed to become worse.

The patient became aware of her lues at age 20 when she developed interstitial keratitis. The mother and brothers were also found to be luetics at this time. Treatment was given. Interstitial keratitis recurred 10 years later. The blood serology has been weakly positive over the years.

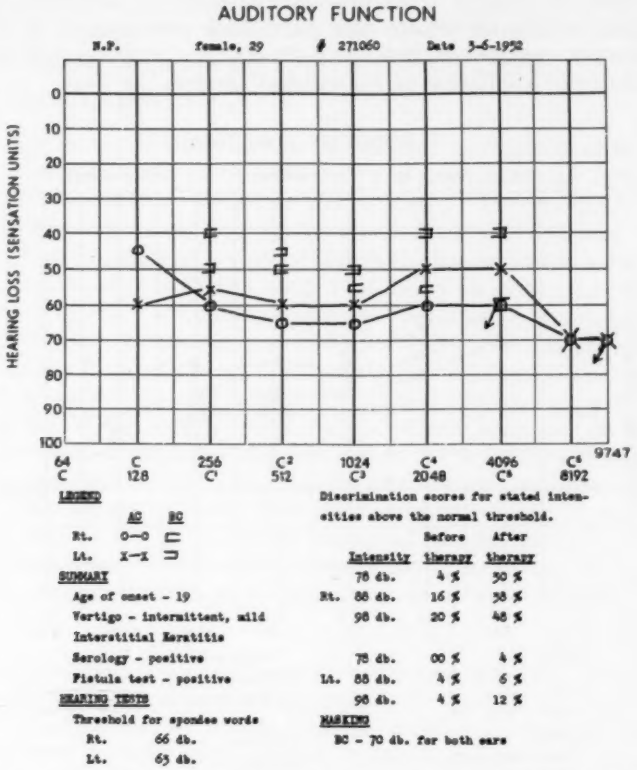

Fig. 9. Audiogram of young woman with 10 years of progressive ear disease due to congenital syphilis that did not respond to extended standard antiluetic therapy over this period. A subsequent course of combined penicillin and ACTH therapy was also without clear-cut effect.

The threshold curves showed a bilateral moderately sloped perceptive loss (see Fig. 10). The cold caloric test showed profound loss on the left and moderate loss on the right ear. Positional changes, especially head shaking, induced vertigo and spontaneous nystagmus to the left. Negative pressure to each ear caused a weakly positive fistula reaction with subjective vertigo.

Comment: The observed histopathology of late congenital syphilis of the ear suggests that changes in the endolymphatic system of the labyrinth, *i.e.*, dilatation of cochlear duct, sec-

ondary to invasion of the labyrinth capsule and perilymphatic space by the spirochete may have some resemblance to the changes reported in Ménière's disease and may account for occasional similarity of the clinical picture.

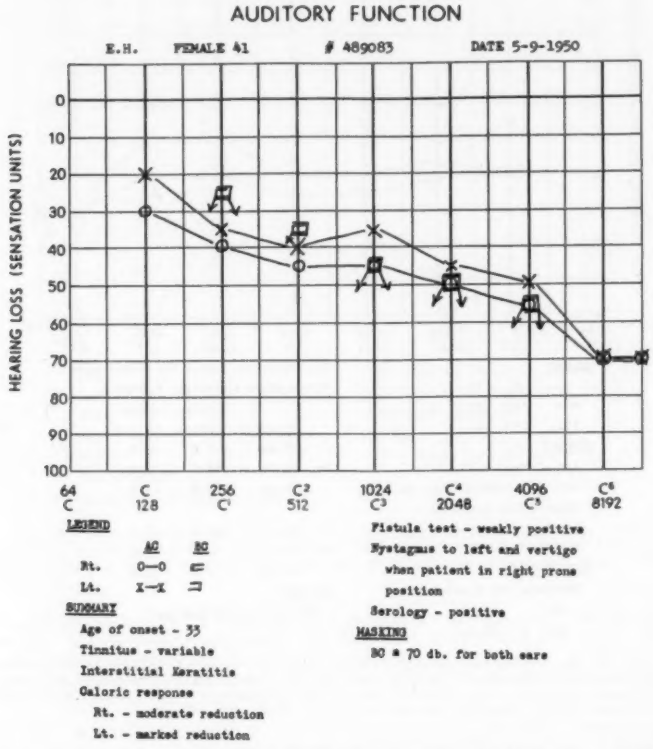

Fig. 10. Audiogram of a patient with eight years of ear disease. An attack of interstitial keratitis led to the diagnosis of congenital syphilis at age 20. Her mother and brothers were found to be luetics at this time. She was extensively treated but keratitis recurred at 33 and ear symptoms began at this time.

Case 11: C. W., male, age 35, was a known congenital luetic with positive serology even after intensive treatment at the clinic for the four years preceding the onset of deafness and tinnitus in the right ear. A year later the patient began to have attacks of vertigo. The hearing test showed a flat nerve loss on the right with a practically normal curve

on the left. The caloric test, however, showed no response to ice water on the normal hearing side and a normal response on the side showing deafness. The fistula test was negative. There was a history of earlier inflammation of the eye, but no evidence of interstitial keratitis was found. There is, however, a suggestion of Hutchinson's teeth. The mother was a known luetic.

Comment: While this case is included in this report as a known congenital luetic with auditory and vestibular symptoms, the clinical picture is not typical of the group. The nature of the right ear deafness and dizziness beginning in the fourth decade is typical of Ménière's disease and may have no relation to the known congenital lues with continued positive serology. The absence of a fistula response is not diagnostic, since Hennebert reported that the fistula response was negative in the male with late congenital syphilis of the ear. More suggestive of some unusual inner ear disease in this patient is the total absence of the caloric response on the opposite practically normal hearing ear. The absence of positive serology in the spinal fluid in this patient, and in others of this group, indicates that the meningeal change is not a primary factor in this luetic disease of the ear which begins primarily as a gummatous osteomyelitis of the temporal bone. The major findings in all the above cases are indicated in Table 1.

Discussion: Like otosclerosis, this disease might be present in the temporal bone for some time before symptoms become manifest. Goodhill[6] found histopathological changes in the temporal bones of luetic infants. In those who survive, lesions may remain dormant and asymptomatic for years, but finally with invasion of the labyrinth capsule, modiolus or ossicular chain symptoms appear and progress with varying rapidity.

The extremely rapid progression of the deafness seen in some of these cases may be due to the widespread serous labyrinthitis initiated when the inflammatory process finally extends through the labyrinth capsule into the labyrinth spaces.

Invasion of the ganglion and nerves in the modiolus may be another important factor in determining the particular course of the disease.

TABLE 1. FINDINGS IN THE PATIENTS DESCRIBED.

	Family History	Serology	Interstitial Keratitis	Teeth	Age of Onset
1. agk	?	—	+	+	11
2. cb	+	+	+	+	17
3. bf	?	+	+	—	18
4. rl	+	—	+	+	17
5. ct	?	+	+	—	35
6. va	+	+	+	—	23
7. mb	?	—	+	—	9
8. ac	+	—	+	—	12
9. np	+	+	+	—	19
10. eh	+	+	+	—	33
11. cw	+	+	—	+	31

	Tinnitus	Vertigo	Fistula Test Pressure: Positive	Fistula Test Pressure: Negative	Caloric: Normal	Caloric: Abnormal	Effect of Pregnancy
1. agk	—	—	—	+		+	—
2. cb	+	+	—	+	+		+
3. bf	+	—	+	+		+	+
4. rl	+	+	—	+		+	+
5. ct	+	+	—	+		+	+
6. va	+	+	+	+		+	—
7. mb	+	+		?		+	
8. ac	+	+	—	?		+	
9. np	—	—	—	+		+	+
10. eh	+	+		+		+	—
11. cw	+	+	—	—		+	

Despite the widespread striking histological changes in the temporal bone demonstrated by Mayer and Fraser, careful X-ray studies have failed to demonstrate them. Spinal fluid serology has been negative in all these cases, confirming their opinion that meningeal activity was not an important factor in the disease.

The high incidence of this disease in the female is definite, but difficult to explain, as is the aggravation of the symptoms by pregnancy. The particular metabolic changes and endocrine imbalance associated with pregnancy sometimes leads to aggravation of chronic systemic infections. Clinical observation indicates that local infections like chronic sinusitis or

otitis media may become more active and severe during pregnancy. The osteomyelitic gumma in the temporal bone and the labyrinth inflammation may thus be activated by pregnancy. Since clinical observations indicate that infections may be more severe while patients are under ACTH therapy, the presence of additional ACTH in the blood during pregnancy may be related to the observed progression of the luetic ear diseases at this time. The question of advising against pregnancy even during the inactive stages of this ear disease is worthy of consideration. When the patient is seen during pregnancy it may be advisable to give intensive antiluetic treatment, particularly with the signs of reactivation of the infection.

Vestibular symptoms as well as abnormalities of vestibular tests are the result of the many degrees of inflammatory changes in the vestibular part of the labyrinth so carefully described by Mayer and Fraser. The positive fistula response with an intact drum is a unique feature of this disease and is related to the observed erosions of the capsule bone in the vestibular labyrinth and oval window. The ossicular chain probably transmits the pressure in the fistula test and this may explain the marked difference between positive and negative pressure in producing nystagmus. Often there is no response at all even with very strong positive pressure. The greater sensitivity to negative pressure is in line with the greater sensitivity of the ossicular chain to displacement by negative static and acoustic pressure accentuated at the stapes. The short duration of the induced nystagmus indicates difficulty in maintaining deviation of the cupula by movement of the stapes (see Fig. 11). Tammari reports that the fistula test was negative in all eight of his cases, while the majority of our patients gave a positive fistula response when negative pressure was used.

The rational of ACTH therapy combined with known antiluetic drugs, is the observation that ACTH tends to reduce fibroblast and connective tissue reaction at an inflammatory site. The widespread inflammatory reaction within the laby-

rinth might be diminished and a similar effect on the luetic lesions in the temporal bone may tend to facilitate approach of the therapeutic agent in the blood stream. Any reduction of this connective tissue barrier to the gummatous foci may reactivate them but this is controlled by the simultaneous administration of the antiluetic drug. Mayer[7] believed that

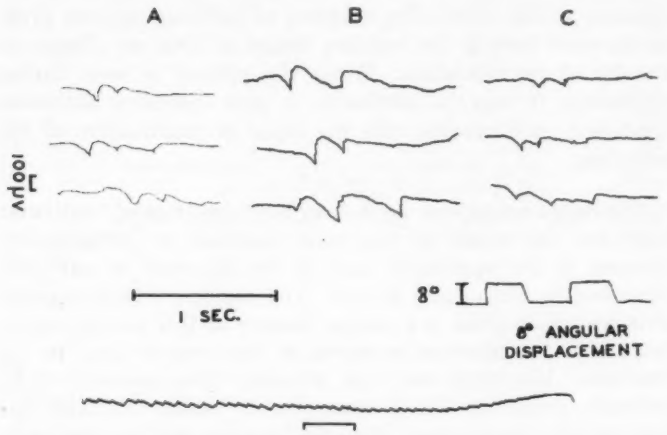

Fig. 11. Record of eye movement induced only by maintained negative pressure in the external canal in the case (NP) of late congenital syphilis of the ear. (A) Eyes open. (B) Eyes closed. (C) Eyes open but covered with Bartel's glasses.

Note that for each stimulus about three eye movements are produced, lasting altogether about one second. The movements are larger when the eyes are closed. Compare with record of voluntary eight degree displacement—lower right. More than 10 times as many eye movements lasting about 10 seconds are produced by similar pressure on a fistula of the horizontal canal in chronic mastoiditis (see lowest tracing. Note: different calibration). Note: Records made with the cooperation of Dr. T. Case.

the failure of ordinary antiluetic treatment in this disease is due to the spirochetes being buried in the inactive capsule and he proposed to reactivate this bone and thus mobilize the spirochete so that it might be attacked by the antiluetic drug. He cited the experiments of others who produced changes in the labyrinth capsule bone and he proposed a low calcium diet free of vitamin D together with salvarsan as a method of treatment of late congenital syphilis of the ear.

The series of cases here reported receiving combined therapy are of course altogether too limited and the duration of observation too short to permit any conclusions. This disease is so serious, however (complete loss of cochlear and vestibular function may occur), and so resistant to standard therapy that an early test with the combined ACTH and penicillin treatment should be considered. Under the combined therapy none of the patients showed further deterioration. Some were so severely deafened that irreversible pathology was probably present which could not be affected by treatment.

SUMMARY.

Eleven cases of congenital syphilis that developed ear symptoms were studied. This ear disease affects young women particularly and is often rapidly accelerated by pregnancy.

The auditory and vestibular symptoms and the results of functional tests are compatible with the bone and labyrinth pathology described by Mayer and Fraser.

A positive fistula test in the presence of an intact normal drum is a distinctive sign in this disease. The response is obtained particularly with negative pressure in the external canal. Unlike the usual fistula response, the duration of the induced nystagmus in these cases is usually very brief. There are about three eye movements lasting about one second.

This ear disease is resistant to antiluetic therapy and may even progress rapidly to profound loss of function during intensive treatment. From a consideration of the special pathology and observations on a few of the patients studied in this report, treatment with penicillin and ACTH when given in early stages appears to offer some prospect for improvement.

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MASKING AND FATIGUE EFFECT OF WHITE NOISE IN CONNECTION WITH SPEECH TESTS.*

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The masking effect of noise on the perception of speech has been amply and accurately covered by many authors; with reference to the rational and appropriate employment of the type of masking tone used, less information is available, as most authors in their experiments used the masking tone normally found in ordinary audiometers; this generally comprises a tone spectrum in which low frequencies predominate.

The introduction of white noise undoubtedly presents a progress in that it enables a masking tone to be used, covering with equal distribution the whole frequency spectrum. It would be well to remember that the term "white noise" or "thermal noise" is derived from the fact that the thermal agitation of electrons in a resistance gives rise to a variable potential across the ends of the resistance itself; the variations are distributed uniformly to cover the frequency spectrum. For our experiments we used an Elit white noise generator, supplying a maximum output of approximately 10 mW. with a 600 ohm resistance. The power supplied by this generator is uniformly distributed between 40 and 10,000 c.s. within limits of ± 4 db, as measured with a General Radio wave analyzer (see Fig. 1).

Using the above apparatus, it was our intention to study the masking and fatigue effect of white noise, no longer using headphones and monaural reception as had been used previ-

*From the Otorhinolaryngological Clinic, University of Milan (Director Prof. L. Pietrantoni).

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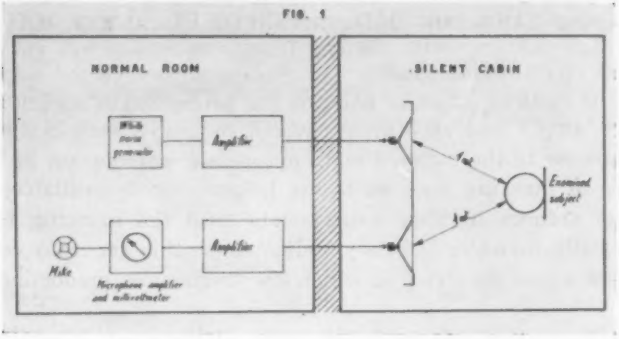
ously by Hawkins and Stevens, Egan, Pollack, but with a loudspeaker acting in free field and using logatomes as test material.*



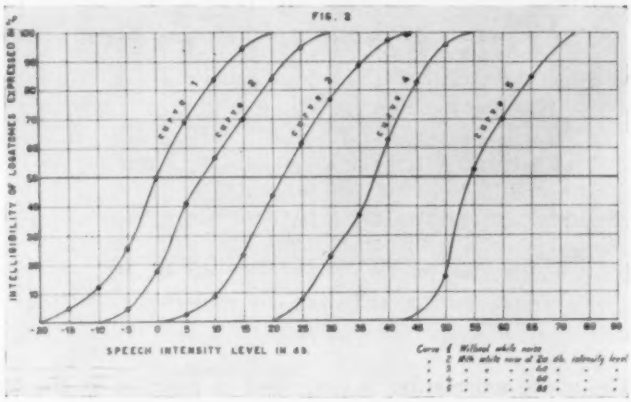
Our research refers to 12 selected subjects with normal hearing. The following research method was adopted: the subject was made to enter a silent cabin and to sit one meter from the two loudspeakers placed one above the other (one emitted white noise while the other transmitted logatomes); the examiner, outside the silent cabin, was thus able to carry out the articulation curve for logatomes in silent conditions. By switching on the white noise generator, he was able to repeat the articulation curves at the following fixed intensity levels of white noise: 20, 40, 60, 80 db above normal threshold.

Having finished this first part of the test and under identical conditions as previously, the subject being examined was required to listen to the white noise signal strongly amplified (in some cases 60 db, in others 80 db) for 15 minutes with the intention of introducing a state of fatigue, after which

*As phonetical materials were chosen, the logatomes, constituting an ideal phonetic material, where research assumes an essential clinical character, as they are a suitable stimulus to the ear only, without interesting superior psychical centers and without identification of the stimulus involving a definite mental engagement. Italian is one of the few languages in the world allowing the use of certain logatomes without subjects being requested to know the so-called phonetic alphabet. In the Italian language there is indeed a rather constant correspondence between some graphical signs and the relating phonetic sounds.

the articulation curve was repeated once more in the presence of white noise at the same fatiguing level, and yet again immediately after the noise had been switched off.

We will now consider the results obtained during the first part of our research, *i.e.*, those dealing with the raising of the perception threshold for logatomes in the presence of white noise at varying intensities (in other words, the "threshold shift"), which has already been studied by Hawkins and Stevens for pure tones and for connected discourse in the presence of white noise and, in Italy, by Azzi, in the presence of the masking tone of a normal audiometer (see Fig. 2).



Studying this graph we can observe that the threshold shift is much lower for low intensities of noise (7 db rise in threshold of perception for logatomes in the presence of white noise at a 20 db level; 21 db rise for noise at a 40 db level), while for higher intensity of noise the threshold of perception for logatomes is subjected to a further rise almost parallel to the increase in intensity of the noise itself (38 db for 60 db level of white noise and 55 db for 80 db level of white noise).

The above results signify that for low levels of masking noise the ear retains sufficient "resolutive power" to oppose the masking effect of the noise itself, while for higher noise levels the masking effect is almost complete.

The results show, therefore, that the masking effect on iogatomes does not proceed in a strictly linear fashion as in the case of pure tone in the presence of the masking tone of a normal audiometer, but according to a curve which initially takes the form of a parabola, while the latter tends to run parallel to the straight curve representing the behavior of the phenomenon for pure tones. This is clearly demonstrated in Graph 3.

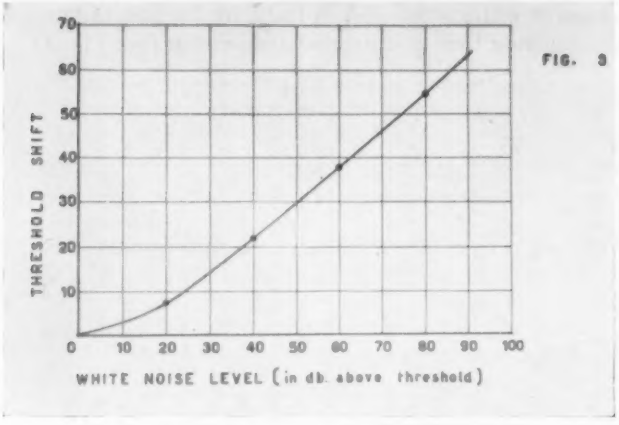


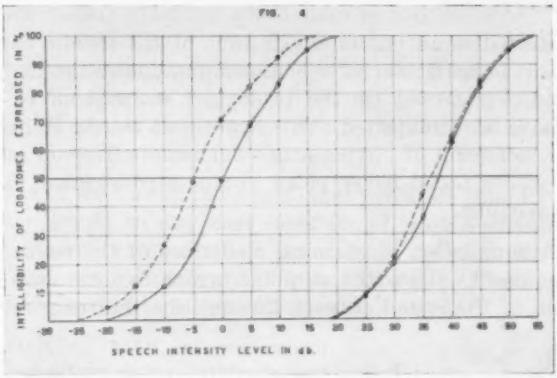
FIG. 3

The above affirmation is confirmed in practice by the fact that long distance telephone conversation can be affected in the presence of low background noise level, the noise itself not having sufficient masking effect to impede speech perception.

Naturally the resolutive power of the ear is not equal for all subjects with normal hearing; for this reason the threshold shift in the presence of white noise can be recommended as a useful test when selecting personnel for certain occupations where good discriminatory faculties are of the maximum importance (telephone operators, radiotelephone operators, echo-sounding operators, etc.). We are here referring, however, to a test which is of interest not only to the otologist but also to the psychologist, since the results obtained depend

also upon the phychological factor, the mental response, and in some degree also upon the standard of education of the subject examined.

The second part of our research, that dealing with the fatiguing effect of white noise, has provided us with real surprises: the articulation curve repeated after 15 minutes of continuous white noise at a high intensity level (in some cases at the 60 db, in others at the 80 db level) proved to coincide perfectly with the curve taken in the presence of white noise at the same intensity level before the fatiguing test took place, thus showing that exposure to white noise at the levels used and for the period of time mentioned produces no fatiguing effect whatsoever (see Graph 4).



The articulation curve taken immediately after the cessation of white noise has shown that where we expected to find a rise in threshold of perception, we found instead a slight lowering of the threshold itself, almost as though the white noise had a "sensitizing" effect rather than a "fatiguing" effect.

Egan had already found a similar phenomenon but in different experimental conditions, *i.e.*, with binaural headphones. The subject being examined was made to listen to white noise

at a low level of intensity (45 to 50 db) through one of the phones, while the other was used to transmit connected discourse. The A. found that when introducing white noise in a phone, perception of connected discourse in the opposite ear was some 5 db better. The hypothetical explanation was that the phenomenon was due either to the reflexed contraction of the middle ear muscles of the opposite ear, or else to the fact that as white noise contains all the frequency bands found in common speech, these bands act to reinforce the intensity of the speech fed to the opposite ear. It should be noted, however, that the results reported by Egan were later refuted by Pollack.

In conclusion, the hypotheses which can be advanced to explain the phenomenon observed by ourselves can be listed as follows:

1. Reduction of the muscular tone of the middle ear muscles, and in particular of the stapedium, due to the fatigue of the muscles caused by the prolonged stimulation to which they have been subjected. This hypothesis should be linked to the phenomenon of "hyperacusis dolorosa" observed in many cases where facial nerve palsy produces consequent palsy of the stapedium.

2. Accumulation of chemical mediators of the nervous system, caused by the sound stimulus, which facilitate the transmission of the sound current through the interneuronic synapsis.

3. Psychic effect due to the absolute and sudden silence following a condition of high intensity prolonged noise, causing the subject better to perceive speech, both subjectively and objectively.

We are not in possession of experimental data to confirm the first two hypotheses. All subjects examined spontaneously observed that they had the subjective sensation of "hearing better"; what is more important, however, is that their observations are not only subjective but objective and clearly demonstrated by Fig. 4. Neither is it possible to raise the objection that they heard better because of their increasing

familiarity with logatomes used throughout the test; first, because our subjects were selected from persons with a good standard of intelligence, who, in the majority of cases, were already familiar with logatomes; second, because the same subject was never presented twice with the same list of logatomes (10 per list). We were in possession of numerous phonetically balanced and, therefore, interchangeable lists.

Another objection which could be raised is that if white noise at 80 db intensity level gave rise to fatigue phenomenon, such fatigue could not, in practice, be prolonged over 30 to 35 seconds starting from the moment when the noise itself ceased, while a complete articulation curve takes at least 60 to 70 seconds to complete. To these objections we can reply as follows:

1. Immediately after the white noise ceased, we started taking the articulation curve, beginning at the value of intensity which we had found to correspond with the threshold of perception at the start of the test. This enabled us to bring out eventual differences between the two perception thresholds in less than 15 to 20 seconds.

2. Even if we were unable to comply with the time limit as above, we would in any case have found, in the absence of fatigue phenomenon, complete coincidence between the two curves, and not that slight, definite and constant difference which we observed in all our 12 cases.

To conclude briefly, the real reason producing the phenomenon observed by us is still unrecognized and for this reason we limit ourselves to reporting it. Further physiological and, above all, physiopathological studies of hearing might give us a better explanation; for this reason we propose to study how the phenomenon and also how the threshold shift, so far observed to be present in subjects with normal hearing, behave in cases of otosclerosis and in nerve deafness.

SUMMARY.

The authors determined the behavior of threshold shift produced by white noise in speech tests with logatomes. After

having noted that such shift is proportionately greater for high intensities of white noise, they described the particular phenomenon whereby the ear gains in sensitivity due to white noise. They indicate some explanatory hypothesis of the above phenomenon.

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AIR EMBOLISM DURING LAVAGE OF THE MAXILLARY SINUS. A REPORT OF TWO CASES.*

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Although the lavage of the maxillary sinus is a simple routine diagnostic and therapeutic procedure, complications can and do occur at infrequent intervals. The most serious and most terrifying complication of this procedure is an air embolism. The suddenness of onset, the terrifying train of symptoms and events that follow, the utter helplessness that the otolaryngologist feels and the serious consequences that may follow produce a picture that is unforgettable. To have a relatively healthy individual walk into the office for this simple procedure and then to have a person carried out on a stretcher either dead or unconscious magnifies the gravity of the situation, and justly so. This makes it all the more important that we examine all the factors and ask ourselves why this complication occurs and what can be done to prevent it. Certainly, it is our duty and an obligation to our patients that we make this procedure completely safe, so that their health and their lives will be safeguarded.

Grove,[1] in his excellent article, gives a historical resumé of the introduction of the lavage of the maxillary sinus through the various routes. It was Copedepont who, in 1894, was the first to introduce the injection of air into the maxillary sinus. Since then it was and still is the practice of many otolaryngologists to insufflate air into the maxillary sinus. Some injected air into the antrum to determine whether the needlepoint is within the maxillary sinus. If the needlepoint is in the sinus, a peculiar gurgling sound of air entering the sinus was supposed to be heard. After the irrigating fluid,

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air was insufflated into the sinus to force out the fluid and whatever mucus plugs are present. The majority of otolaryngologists do not inject air before the irrigating fluid, but many do so after, in order to dry out the sinus. This practice of injecting air into the antrum has led to a number of very serious and even fatal complications.

It was Hajek[2] who, in 1907 at the German Laryngological Society, was the first to refer to this very serious complication which he observed during lavage of the maxillary sinus. He attributed this to an irritation of the vagus, cocaine poisoning, hysteroepilepsy and epilepsy. From then on, numerous similar cases were reported, some of which led to death and others to recovery. All of these had the same train of symptoms, which were characterized by a sudden collapse into unconsciousness, cyanosis, pulse and respiratory changes, tonic and clonic contractures of various groups of muscles and indeed, all muscles, and muscular twitchings. No logical explanation was given for this complication and the cause was variously reported to have been due to cocaine poisoning, or a nasal reflex neurosis which was due to an irritation of the vagus through the trigeminal nerve in the nose.

Bowen,[3] of Toronto, in 1913, reported two cases of air embolism following antral lavages. In the first case, a female became unconscious and had convulsions following a puncture of the antrum and the inflation of air. She recovered after being unconscious for five days. The second case, a male had a puncture of the antrum and immediately after the insufflation of air became unconscious, had convulsions and died within an hour. At autopsy, the heart was markedly distended. A hissing sound was heard when the pulmonary artery was opened and the distended heart collapsed. The puncture of the antral wall was easily found and when the anterior wall was removed, the mucous membrane of the roof, except for a small part, as well as the outer and inner walls was found detached. The mucous membrane covering the roof was hanging about a quarter of an inch below, and it showed a small puncture mark. The bone of the roof and posterior wall did not show any puncture mark. Consequent-

ly, the needle did not puncture beyond the antrum. Bowen postulated that the needle punctured the mucous membrane of the roof, and the air was forced between the mucous membrane and bone, opening up numerous capillaries, thus allowing the air to pass into the general circulation.

Boenninghaus[4] and Gording,[5] in 1920, were the first to point out that the cause of these serious symptoms was an air embolism. Boenninghaus found that all these cases had one thing in common: the symptoms usually followed injection of air into the maxillary sinus. He expressed himself as not believing that these mishaps were due to cocaine and novocaine poisoning. While he did not deny that vagus excitation may arise from too strong air pressure or too cold an irrigating fluid, he did not believe it possible for this factor to function to such a degree as to produce an actual death. He reported 11 fatal cases in which autopsies were performed and concluded that the air reaches the right heart through the facial veins by the internal jugular vein, but that it may also cause death by hitting the breathing center in the medulla.

Gording,[5] in 1920, collected nine cases from the literature and reported four of his personal cases and another three related to him by his colleagues. There was one death in his own four cases. He felt that the cause of the trouble was an air embolism and was able to prove this experimentally in rabbits. By injecting air into the maxillary sinus of the rabbits, he could produce changes in the respiration down to complete cessation although no changes in the pulse were noted. In one of his rabbits, he produced respiratory changes on irrigating one antrum, but on irrigating the other side convulsions and death ensued. At autopsy it was seen that an incomplete puncture had been done and that the mucosa of the sinus had been stripped from the nasal wall, occasioning bleeding. There were air emboli in the right heart and in the general circulation. He concluded that in thick walled antra where the puncture is made with difficulty, it is possible to loosen the mucous membrane from the underlying bone and open up the blood vessels. In these cases, the mucosa is

pushed ahead of the needle and on inflation air is then directly forced into the circulation; therefore, he assumed that in the cases where the bony wall was thick and collapse came on immediately after inflation, the cause was air embolism. In the other cases where the bony wall was thin and the symptoms did not come on until actual syringing began, he assumed that a trigeminal reflex could be the cause of the catastrophe. Grove[1] does not believe that this latter point is correct because with the syringe used, very little time is lost between the cessation of the inflation and the beginning of the irrigations, and it might easily take the small interval of time for air which had entered the circulation during the inflation to reach the coronaries or vessels of the medulla.

Grove,[1] in 1922, made a most complete and exhaustive study of the subject. He was able to collect 42 cases of air embolism following antral lavage. Of these cases, there were 15 deaths, 11 of which occurred immediately or within a few hours to 36 hours. In these fifteen cases, seven followed a puncture through the inferior meatus, three from the middle meatus, while in five cases the route was not mentioned. In the autopsies done on eight of these cases, three were entirely negative; in two cases minute hemorrhages or signs of stasis were found in the circulation. In a second group of 25 cases the chain of symptoms was the same except for the fact that the patients all recovered after a harrowing experience. In these cases the puncture was done through the inferior meatus in 15 cases; through the middle meatus in two cases, and in eight cases the route was not mentioned. In the next six cases the common symptom was transitory blindness. The most classical example of this was the case cited by Caesar Hirsch,[6] of a 16-year-old girl who, after lavage of the antrum, complained of a sudden inability to see out of one eye. The pupil in that eye was immobile, but no pain was complained of. An opththalmoscopic examination revealed a pale optic disc, grayish retina and normal retinal veins. In the arteries, bubbles of air were plainly visible, separated from each other by columns of blood slowly moving. After two or three minutes, the air left the arteries; the retina assumed a more

normal color and the disc lost its distinct pallor. The activity of the pupil and the sight also returned. In these six cases, two followed a puncture through the inferior meatus, three from the middle meatus and in one the route was not mentioned.

Since writing his paper Grove's attention was called to two more cases of collapse during inflation from the practice of two of his colleagues and one case from the literature. J. C. Coffey,[7] of Milwaukee, reported a case of a 54-year-old female in whom, after cocainization, a puncture of the right antrum through the inferior meatus and an irrigation of normal saline was done without incident. The procedure was repeated on the other side with the same anesthesia. Considerable resistance was met in attempting to force the water into the antrum, and this caused the patient some pain. At the same time, only very little water returned. The patient collapsed, became cyanotic and unconscious, with convulsive movements. Death seemed imminent, but the patient recovered after half an hour.

Richard Meunzer,[8] of Milwaukee, reported a case of an 18-year-old woman who had a puncture of the left antrum, air inflation and then was irrigated with normal saline. Upon drying out the cavity with air, the patient suddenly collapsed and became dyspneic and had twitchings of the muscles. She recovered in two hours. A second puncture repeated a week later produced the same train of symptoms.

H. L. Lanier[9] reported a case of a 52-year-old female who had a puncture of the left antrum through the inferior meatus under 10 per cent cocaine anesthesia. The puncture was done quickly and easily, and the first few injections of air passed through the antrum without resistance. Several compressions of the bulb were made in rapid succession when a mucopurulent mass was blown out through the maxillary ostium. After the ostium again appeared to be blocked, more air was insufflated. The patient fell prostrate, became cyanotic, the pulse was small and hardly perceptible and the respirations became irregular and shallow. The patient remained in this state for three hours and then regained consciousness.

Since Grove's article there has been a paucity of reports in the literature. Bacher,[10] in 1923, reported a fatal case with autopsy findings corroborating the results. Cavanaugh,[11] in 1925, reported a case of a 35-year-old female who developed a hemiplegia following lavage through the natural ostium and injection of air into the antrum. The patient remained unconscious for four weeks and then gradually improved and eventually recovered. Goldenburg,[12] in discussing Cavanaugh's paper, mentioned a case of a 35-year-old female who died an hour after a puncture of the antrum and the insufflation of air under pressure. An autopsy revealed an air embolism in the heart, and in the coronary, cerebral and mesenteric vessels. Lebensohn,[13] also in discussing Cavanaugh's paper, knew of one case where even the air in the tube connecting a Lichwicz needle with a pint piston syringe was sufficient to cause a temporary hemiplegia from which the patient recovered in a few hours.

McGovern,[14] in 1941, reported two cases with one fatality. The first case, a 24-year-old Negro male, had a puncture of the antrum through the inferior meatus. After the injection of air into the antrum, he became unconscious, developed severe convulsions and died. An autopsy was not performed. In the second case, a 24-year-old white female with diabetes had the antrum lavaged through the natural ostium. Upon the inflation of air, she became unconscious, and had severe convulsions. She subsequently recovered in one week.

Bashir, Taneja and Shah,[15] in 1945 described a case of cerebral embolism following the puncture of the antrum and the injection of air. The patient collapsed, developed convulsions and paralysis, but subsequently recovered completely.

Cornfield and Laszlo,[16] in 1949, reported a death following antral puncture and the insufflation of air. The patient, a 46-year-old white male, collapsed and was seized with generalized convulsions, ceased breathing and died in spite of artificial respiration, and cardiac and respiratory stimulants given intravenously and into the heart. An autopsy performed six hours later revealed a markedly distended heart

and vena cava. There was a loud audible release of air when the heart was opened. No air was found in the cerebral vessels. The cause of death was reported as due to a massive air embolism.

F. J. Pinkerton,[17] of Honolulu, has had two fatal cases in his experience. In the first case, a male had a large polyp which protruded from the antrum and a purulent maxillary sinusitis. The polyp was removed. Three weeks later, a lavage of the antrum was done through the natural ostium. The cannula was introduced without difficulty; in fact the cannula "practically fell in" as the operation had left the ostium wide open. During the lavage with the fluid, there was some blood. Upon the insufflaton of air, the patient fell unconscious, had a convulsive seizure and died within 20 minutes. An autopsy revealed air embolism. Pinkerton feels that in this case, the lavage may have loosened a few blood clots and left open the ends of a few blood vessels for the entrance of the air. In the second case, a male with purulent maxillary sinusitis had repeated lavages through the natural ostium for a purulent infection. In what was supposed to be his last lavage, he fell unconscious after the lavage and the insufflation of air, had convulsions and died within half an hour. An autopsy revealed air embolism.

Kelley[18] reported an interesting case of a woman on whom he performed a puncture of the antrum through the inferior meatus. On insufflation of air, a low gurgling sound was heard unmistakenly passing down the neck. The patient stated that she felt something passing down the neck but was in no way affected by it. Kelley felt that the needle must have passed through both walls of the antrum and opened up a vessel in the pterygoid plexus of veins in the pterygomaxillary fossa and thus air entered into the circulatiin.

There is no doubt regarding the manner of development of air embolism. The air must get into the circulation in only one way and that is, during the puncture, a vessel must be punctured or injured and the air is forced directly into the circulation.

Gurlitz[19] has shown that the antral mucosa is very vascular and in 50 per cent of his injected specimens there was a thick venous plexus over the antral wall or orbital wall, or over both walls, that whenever this plexus is present it is usually where it can be easily injured by the puncture. Gurlitz's specimens were those of normal sinuses; moreover, in the infected maxillary sinuses where the antral mucosa is thickened and congested and frequently almost fills the maxillary antrum with loose edematous folds, one would expect to find an increase in the number of blood vessels.

Gording[5] has studied very carefully the mechanism of the development of air embolism. According to him, if the needle is advanced at a slow rate through a thick wall of the inferior meatus, the mucous membrane may be detached over a considerable area, that the blood vessels may be torn in these cases, the air is forced into the torn blood vessels and air embolism results. In Bowen's[3] case, the mucosa of the roof, except for a small part as well as the outer and inner walls, was found detached as a result of a puncture through the mucous membrane of the roof and the forcing of air between the bone and the mucous membrane.

After the air has entered the vein, it then proceeds from there to the right chamber of the heart. From here, opinions in regard to the cause of death differ greatly. Some consider that it is a "lung death" and believe that the air is driven from the right chamber of the heart into the lung capillaries which are embolized and the circulation is interrupted, with death as a result. Most writers are inclined to the view that the cause of death should be looked for in a paralysis of the heart and it should be regarded as a "heart death." In many cases, the predominant symptoms are nervous disturbances. Some workers assume that the air bubbles have passed through the lesser circulation and entered the arterial system, where they caused, through emboli of the brain vessels and medulla oblongata, the periods of unconsciousness, the convulsions and respiratory disturbances, thereby causing a "brain death."

Bergstrand,[20] basing his opinion on his rabbit experiments and clinical observations, felt that the danger with venous air embolism is from a drop in the arterial pressure as the accumulating bubbles of air reach the pulmonary capillaries. The slowing of the current raises the pressure in the aortic vessels. This causes an anemia of the brain with neurological symptoms and death. He found that paresis, indicating a local injury of the brain, was never noted in the animals or in clinical cases. The blood in the aorta is a dark color, indicating a defective oxygenation; hence inhalation of oxygen seems a rational treatment. Also, by lowering the head the negative hydrostatic pressure of the veins tends to prevent any inflow of air. Even after the low pressure has been combatted, the danger is not past as the air accumulates in the right heart. He has never witnessed any benefit from a puncture of the right ventricle in the animals as the air escapes into the pulmonary artery and is not released by puncture. Venesection may give relief with any form of air embolism. With arterial air embolism, he found that the air enters the left ventricle and passes immediately into the aorta and bubbles are found in the peripheral arteries, especially at the base of the brain. He also witnessed the presence of air bubbles in the choroidal vessels of the eye until they were gradually absorbed or swept along. He estimated that 75 to 100 cc. of air can be borne in a venous embolism without permanent harm, but even 1 cc. of air in the artery is sufficient to kill a horse. It must be assumed, then, that in the cases of severe nervous manifestations and of death, the air must enter the arterial circulation. It has been proven not only that air in the arterial system passes without difficulty through the lung capillaries but also that air bubbles in the venous circulation may be carried into the arterial circulation.

Grove, himself, felt that the nervous manifestations would arise during antral inflation if the air pumped into the circulation was insufficient to fill the right heart completely and stop its action suddenly. The air would then be forced

through the lungs and into the left heart and the greater circulation. By passing into the vessels of the brain and the medulla, it would cause symptoms until it was absorbed.

Kelley[18] has called attention to the fact that the grave symptoms which arise from the puncture of the antrum and the insufflation of air are strikingly similar to those which occasionally arise during the production of an artificial pneumothorax.

The symptoms of air embolism are characteristic and once seen are unforgettable. The onset occurs with dramatic suddenness and the patient usually slumps in the chair and becomes rigid and unconscious. The unconsciousness is associated with pulse and respiratory changes, cyanosis and then convulsions. The respirations may be irregular, stertorous, suspended and jerky, with periods of apnea. There may be tonic and clonic contractions of various groups of muscles almost akin to an epileptiform seizure. A severe trismus has been noted in several cases. The pupils may become fixed and dilated, or there may be a nystagmus, conjugate deviation of the eyes, or a backward rolling of the eyeballs. The pulse may be bounding, irregular, rapid or it may even be slow. There often is an associated cyanosis or an ashy gray color. As a rule, there is an elevation of the temperature. Death may ensue immediately or may occur hours or days later. Those that survive the initial onset may have transient disturbances of vision, alternating periods of blindness, varying degrees of paresis, paralysis and even hemiplegia, a feeling of fear and suppression, disorientation, mental lapses and other mental symptoms.

Grove[1] reported six cases in which the common symptom was a transitory blindness. Caesar Hirsch[6] feels that in the cases where visual disturbances exist, the air emboli are visible by immediate ophthalmoscopic examination and are recognized by the pallor of the disc, silvery, narrow arteries with air bubbles within their lumina, normal veins and a slightly clouded eyeground. In his case of the 16-year-old girl mentioned earlier, he was able to see the bubbles of air in the retinal arteries.

The signs and symptoms of air embolism are so characteristic that once seen they are unforgettable. The only condition that may possibly confuse the diagnosis is a severe cocaine reaction. In the milder cocaine reactions, there may be a feeling of anxiety, giddiness and fear, motor restlessness, headache, vertigo, hallucinations, extreme pallor and paresthesia. The pulse is accelerated, and the patient may experience palpitation. The pupils are dilated, and there may be nausea and vomiting. In the severe reactions, there is a dilatation of the pupils, dryness of the mucous membrane, vomiting and clouded sensorium. There may be epileptiform convulsive seizures that may be brief or more lasting. Circulatory and respiratory depression may follow and manifest itself by a feeble pulse, slower and irregular respiration, and cold and clammy skin, Cheyne-Stokes respiration and finally collapse and coma may then occur. Death usually results from a paralysis of the vasomotor and respiratory center of the medulla.

Thus it can be seen that only the most severe cocaine reactions may cause a confusion in the diagnosis. Most of the cocaine reactions are preceded by the premonitory symptoms mentioned above, while in the air embolism the onset is dramatic and sudden. The other and most important point is that an air embolism always follows and never precedes air insufflation.

Attempts have been made to determine the frequency of air embolism in relation to the method of lavage of the antrum. Air embolism may follow any one of the three methods of antral irrigation. In Grove's death series of 15 cases, a puncture was done through the inferior meatus in seven cases, through the middle meatus in three cases, and in five cases the route was not mentioned. In the next 22 cases, with symptoms very similar to the death cases, there were 12 punctures through the inferior meatus, two from the middle meatus, and in eight the route was not mentioned. In the six cases of transitory blindness, two followed puncture through the inferior meatus, three from a puncture through the mid-

dle meatus, and in one case the route was not mentioned. The cases of Coffey, Meunzer and Lanier, that were mentioned by Grove, all followed an inferior meatal puncture.

In Bacher's case, the lavage was done through an inferior meatus puncture. Cavanaugh's case followed an irrigation through the natural ostium, while Goldenberg's and Lebensohn's cases were through the inferior meatus. In McGovern's two cases, one followed lavage through the natural ostium while the other followed a puncture through the inferior meatus. The case reported by Bashir, Taneja and Shah resulted from an inferior meatal puncture, as was the case of Cornfield and Laszlo. Pinkerton's two cases followed a lavage through the natural ostium, as did my two cases.

From this series of 58 cases, 30 followed a puncture through the inferior meatus, 14 from the middle meatus, six from the natural ostium, while in eight cases the route was not mentioned; thus it would seem that this complication is more frequent following a puncture through the inferior meatus. Anatomically, this would seem to be borne out by Gerlach's anatomical study which showed that there is a large plexus of veins in the medial wall of the antrum in the region of the points of entrance of the trocar; however, despite these facts, the seeming predominance in the incidence of air embolism following a puncture of the inferior meatus may be due to the fact that this is the route most often used.[26]

Nothing much has been written regarding the treatment of this condition. From all indications, the treatment does not seem to influence the outcome and is entirely symptomatic. Artificial respiration and respiratory stimulants should be given if the respirations stop and circulatory stimulants for cardiac arrest. The inhalation of oxygen is a rational treatment in view of Bergstrand's experimental and clinical findings of a general anemia of the brain resulting from a drop in the aortic pressure and the resulting defective oxygenation. Also by lowering of the head, the negative hydrostatic pressure may tend to prevent any inflow of air.

Bergstrand also mentions the use of venesection. In his rabbit experiments, he had never noticed any benefit from a puncture of the right ventricle.

The prognosis of air embolism is serious. In general, it depends upon the amount of air and the rapidity with which it is pumped into the circulation as to whether the patient is going to die or recover. It also depends upon the organ that is involved. In the 58 cases that I have collected, there were 23 deaths with a resulting mortality of 39.5 per cent. McGovern states that the mortality varies from 15 to 50 per cent, and that the patient surviving the first 15 minutes has a better prognosis. Just how or where he derived these figures is not mentioned in his article.

In many of the cases air embolism was demonstrated at autopsy. Air has been found in the cerebral vessels, the mesenteric vessels, the pulmonary artery, the juguler veins and in the lungs. In a few cases, the right heart was found distended with dilated pulmonary arteries and hissing of air was heard on the opening of the heart and pulmonary veins; however, in many cases, the presence of air was not demonstrated. Grove feels that even in those cases of death in which no air was found in the circulation on autopsy, an air embolism which was overlooked might have been present in the coronary arteries or in the vessels of the brain. It is easy for pathologists to overlook this factor, for few of them think of opening these organs under pressure unless the clinician specifically requests it.

How can we prevent air embolism following a lavage of the antrum? This is a most important question in view of the severity of this condition. Although air embolism is supposed to be relatively rare, it probably is more frequent than is reported because many surgeons are either loathe to report their bad cases or are too busy and lack the time and energy to do so; furthermore, it could be that many cases of air embolism were probably diagnosed as cocaine reactions. In my opinion, the only sure way of preventing this complication is to discontinue entirely the insufflation of air into the maxillary sinuses.

It was formerly the practice of the older otolaryngologists to insufflate air into the antrum before irrigation and again after irrigation to dry out the sinus. This practice of insufflating air before the irrigation to determine whether the point of the needle is in the sinus and whether the ostium is patent, is entirely unnecessary. If the point of the trocar or cannula is within the sinus, it should be freely movable and there should be no pain or resistance during irrigation. If the point of the needle has passed through the nasal and lateral walls of the sinus from the inferior meatus or through the nasal and orbital walls from the middle meatus, its shaft will not be movable. At any rate, should there be any doubt about the correct position of the needle, a small syringe may be used first to withdraw air, pus or mucopus from the sinus before irrigating the field.

It is also unnecessary to dry out the sinus after irrigation, because there is absolutely no harm in leaving a small amount of saline solution, boric acid or any other mildly antiseptic solution in the antra after the gross products of infection have been removed. Normal saline in itself is stimulating to the cilia and any irrigating fluid is rapidly emptied by the ciliary action or by changing the position of the head or by blowing the nose.

There seems to be little or no disagreement among modern day otolaryngologists regarding the insufflation of air into the antrum prior to the irrigating fluid. This practice has been discarded by nearly all otolaryngologists and has been discouraged by all textbooks and the postgraduate schools of otolaryngology; however, there still is a disagreement regarding the question of inflation of air into the antrum after the irrigating fluid. In a review of the textbooks of otolaryngology, it is seen that most authors advise against this practice. Hajek,[2] Morrison,[21] Turner,[22] Lederer[23] and Neivert[24] warn of the danger of air embolism. Lederer further advises the evacuation by suction and then irrigation with normal saline followed by suction evacuation again. Phillips[25] and Van Alyea[26] advise the use of air after irrigation. Van Alyea states that, "Inflation following irrigation removes the resi-

due to the solution and helps to promote early restoration of function. The dangers associated with inflation are contingent upon the presence of the cannula tip in the nasal or sinus mucosa. This is not likely to be the case if the sinus has just been irrigated and its contents removed. When a return flow has been established, one may be sure the tip is not imbedded in the membrane. The injection of air preceding lavage, when there is some doubt as to the exact location of the tip, accomplishes nothing and may carry with it some element of danger. Its use should be discouraged as hazardous and worthless procedure."

There is also a difference of opinion regarding this point in the teachings of the various medical schools which conduct a graudate course in otolaryngology. Schools which teach their students to inflate the sinuses after the irrigating fluid include the University of Pennsylvania,[27] Washington University of St. Louis[28] and New York University.[29] Schools that advise against the inflation of air include Northwestern University,[30] Harvard University[31] and Tulane University.[32]

Personally, having had two unfortunate experiences, I am strongly opposed to the inflation of air into the antrum, especially since I can see no advantage in doing so. During our training and residency days, we were taught to lavage the antrum with normal saline, using an irrigating bottle connected with compressed air. It is quite possible for air to escape before the fluid or to be mingled with the fluid. I am sure that this was the cause of the mishap in my first case. Right or wrong, I have discarded the use of an irrigating bottle and compressed air and have substituted the use of a Pitkin Sana-Lok Syringe. In using this syringe, great care is taken to see that all the air is emptied from the syringe and the connecting tube before any solution is injected into the antrum; furthermore, the nurse who manipulates the syringe is trained to report the least bit of pressure felt while injecting the irrigating fluid.

Pinkerton feels that if any air is to be insufflated, one should be very careful in the recent postsurgical case as the

ends of the small blood vessels may still be open and predispose to an air embolism. Again, why inject any air at all?

The following is a report of two cases of air embolism that have happened in my practice:

Case 1: A. L., a 25-year-old Chinese male, was first seen on March 1, 1940, with the complaint of nasal obstruction, a purulent nasal discharge and rheumatic pains in the joints. An otolaryngological examination revealed a large septal spur on the left side which was jammed up against the left middle meatus, a polypoid degeneration of the posterior tip of the left inferior turbinate and pus in the left middle meatus and nasopharynx. A submucous resection of the nasal septum was done on April 13, 1940. Subsequent to this, he reported periodically for treatment and complained of a continued nasal discharge. On May 21, 1940, a lavage of the left antrum was done through the natural ostium. During the lavage, it was noticed that air was also escaping from the irrigating bottle together with the nomal saline. Suddenly the patient collapsed in the chair, had a carpopedal spasm followed by generalized clonic convulsions; the pulse was rapid but of good quality; and the respirations were jerky and irregular. The pupils were small and reacted to light. Between convulsive seizures, the patient appeared flaccid. After an hour it was decided that this was more than the usual "fainting spell' and the patient was sent to the hospital in an ambulance. While being carried down the steps on a stretcher, he had a severe convulsion, dropped from the stretcher and landed head first on the concrete floor from a level of 12 feet, suffering a laceration of his scalp in the region of the right occipitoparietal area.

On admission to the hospital, the blood presure was 124/90; the temperature 102.4° F., rectally; the pulse 140/min. and the respiration varied between 32 to 40/min. R. B. C., 6,150,000 with 90 per cent hemoglobin; W. B. C., 21,400 with 90 per cent polys. Urinalysis normal. X-ray of the skull was negative for a fracture.

A neurosurgeon was called into consultation and his notes were as follows: "The patient is semiconscious, and will respond to commands. Lies with his eyes open, staring and expressionless. Any attempt to move him causes him to kick violently with his right leg. Respirations are quiet and regular. Occasionally he spits out saliva forcefully; swallows, but keeps mouth tightly closed. Fundi normal. Pupils widely dilated and fixed to light. There is a constant oscillating conjugate movement of the eyes upward and to the left; a type of nystagmoid movements with the quick component toward the midline. This movement is at times a wide excursion and at other times a narrow one. Corneal reflexes are present with the left side more active than the right. He moves all extremities except the right arm, which is moved reluctantly. The right leg, however, is almost constantly kicking, elicited by movement of the arms and legs. The movement of the extremities cause extreme extensor rigidity; arms extended and pronated; fingers over thumbs. No flaccidity. The biceps, triceps, radical reflexes, knee jerks and ankle clonus are slightly hyperactive. The plantar reflexes are diminished and the abdominal reflexes are absent. *Impression: 1.* Possible extradural hemorrhage from skull fracture. 2. Intracortical hemorrhage—spontaneous."

A spinal puncture revealed normal pressure; the fluid was a pinkish-yellow with floating red cells. Microscopic examination showed five leucocytes per c.mm. and numerous blood cells.

The patient continued comatose; in fact, the coma deepened and he had two projectile vomiting spells. He had typical Jacksonian convulsions starting from the left fingers and wrist and gradually extending to the left side of the body. During these seizures, the eyes deviated to the right. After six of these convulsive seizures, which occurred at short intervals, it was decided that there probably was an irritative focus on the left side, probably in the motor areas. The convulsions were not controlled by sodium luminal, so one-half gr. of morphine sulphate was given subcutaneously. Within an hour, the respirations became depressed; he ceased breathing for several minutes but commenced to breathe again after the administration of artificial respiration. He was rushed to surgery, during which time artificial respirations had to be continued in order to keep him alive. A right temporoparietal craniotomy was done. The brain was found to be under great tension and herniated considerably. On aspiration of the fluid from the frontal horn of the left ventricle the fluid appeared to be under great pressure. After aspiration, the patient's respiration improved and he breathed 12 to 14 times per minute by his own effort. No evidence of any cortical hemorrhage was seen. A large subtemporal decompression was made in the lower portion of the bone flap. He was given several blood transfusions and was returned to the ward in fairly good condition.

Postoperatively, his respirations were free and easy and his temperature, though lower than at surgery, continued at 102° and 103° F. His condition was considered satisfactory, but on the second postoperaive day he suddenly became cyanotic and expired in spite of all treatments.

An autopsy revealed cerebral congestion and edema; cerebral extravascular hemorrhage of the left temporal lobe and the left motor cortex; pulmonary congestion; diffuse toxic hepatitis; cloudy swelling of the kidneys and subendocardial hemorrhage.

Case 2: P. L., a 40-year-old Chinese male, was seen at periodic intervals for a right maxillary sinusitis. He was in good health except for the recurrent attacks of sinusitis. He had had a previous submucous resection of the nasal septum and numerous lavages of the antrum, both through the inferior meatus and through the natural ostium, without incident. On July 20, 1945, a lavage of the right antrum was done through the natural ostium and air was insufflated to dry out the antrum. Suddenly the patient gave a grunt, stiffened up and fell over backward in the chair unconscious and stopped breathing. He was immediately placed on the floor, artificial respiration was started and 1 cc. of adrenalin chloride 1:1,000 was injected intravenously. After his respiration returned, it was sterterous and irregular, and there was much foaming of the mouth. There was an alternating spasm and relaxation of the muscles of his extremities and a severe trismus. The pulse was rapid but of good quality. He was cold and clammy, with a profuse perspiration. The pupils were small and reacted to light, but there was a conjugate deviation to the left. The patient was hospitalized and a neurosurgeon was called into consultation.

On admission to the hospital, the temperature was 98.2° F., the blood pressure 130/80, pulse 120, and the respiration varied between 20 to 30 per minute. He was given oxygen through a nasal catheter; the foot of the bed was elevated; penicillin was given intramuscularly, and caffeine sodium benzoate when necessary. He continued in coma and his temperature rose to 103° F. The convulsions continued, and there was a marked extensor spasm of all four extremities intermittently with a clock-like decerebrate rigidity. Between attacks of spasms, the extremi-

ties were flaccid and there was a bilateral ankle clonus. The pupils became dilated but reacted to light, and there was a continued conjugate deviation to the left. The patient's condition became worse, with a gradual climbing of the temperature, a more irregular respiration until there was a Cheyne-Stokes respiration. The stomach became markedly distended, and a Wagenstein suction bottle was used to attempt to control the gastric dilation. The tracheobronchial tree began rapidly to fill up with mucus, so an attempt was made to aspirate the trachea. As this was being done, the patient developed a reverse peristalsis and vomited up an enormous quantity of dirty black material and ceased breathing.

At autopsy, there was edema of all the lobes of the lungs and patchy areas of consolidation of both lower lobes. The trachea and primary bronchi contained a small amount of clotted blood. The heart appeared normal but was not opened under water. The stomach was markedly dilated and contained much dark, thready material which had the appearance of old blood. The rest of the gastrointestinal tract was normal. The left maxillary sinus revealed a purulent infection and a small polyp. Sections through the brain showed purplish-gray discolored areas at the level of the optic chasm, the anterior end of the third ventricle, at the tip of the globus pallida on the left side and the lower part of the midpons. The area in the midpons was 1 cm. in diameter and appeared to encroach upon the pyrimidal tract. There was no gross lesion to be seen, such as hemorrhages, softening or massive air embolism.

Comment: In the first case, the picture was complicated by the unfortunate fall; however, the clinical picture before the fall was typical and characteristic of an air embolism. The findings at autopsy were complicated and were overshadowed by the cerebral edema subsequent to the injury and the surgical procedure.

In the second case, the symptoms also were characteristic and typical of an air embolism, although no air embolism was found. At autopsy, however, the heart and pulmonary artery were not opened under water.

SUMMARY.

1. The most serious and alarming complication of a lavage of the maxillary sinus is an air embolism.

2. A review of the literature is presented. Fifty-eight cases of air embolism have been collected. Of these 58 cases, there were 23 deaths with a resulting mortality of 39.5 per cent.

3. The signs and symptoms of air embolism are characteristic and once seen are unforgettable.

4. Two cases of air embolism following the lavage of the antrum and the insufflation of air are presented.

CONCLUSIONS.

1. The inflation of air following a lavage of the maxillary sinus is not only unnecessary but exceedingly dangerous.

2. The only way to prevent this complication is to avoid the insufflation of air into the antrum.

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BRONCHIAL ADENOMA.*

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Bronxville, N. Y.

Sixty-nine years ago Mueller[1] reported the first bronchial adenoma. At necropsy on a woman who died from suppurative lung disease of eight years' duration, he found a pedunculated adenoma partially occluding the left main bronchus.

In 1927, Heine[2] found a polypoid tumor the size of a grape in the right middle lobe bronchus of a 45-year-old woman. It had given rise to no symptoms, although bronchiectasis was noted distally to the tumor.

A year later, Reisner[3] reported the death of a woman from empyema and bronchial obstruction. This obstruction was due to an adenoma. In 46 years only three cases of bronchial adenoma were reported, and all were diagnosed at necropsy.

Since the last report in 1928 many excellent articles relating to the various aspects of bronchial adenomas have appeared. Prior to 1944 were the reports of Womack and Graham,[4] Brun and Goldman,[5] Foster-Carter,[6] Goldman and Stephens,[7] Anderson,[8] Adams *et al.*,[25] Laff and Neubuerger,[9] and Riordan and Richards.[10] Since 1944 are those of Jackson, Konzelmann and Norris,[11] Nager,[12] Graham and Womack,[13] Chamberlain and Gordon,[14] Harris,[15] Clerf,[16] Souders and Kingsley,[17] Moersch and McDonald,[18] Maier and Fischer,[19] McCart,[21] Holley,[23] and Huizinga and Iwema.[27] In all, about 300 cases have been tabulated, nearly all of which were diagnosed antemortem.

Almost all early writers stressed the benignancy of these growths, although Kramer,[20] in 1930, suggested that local ma-

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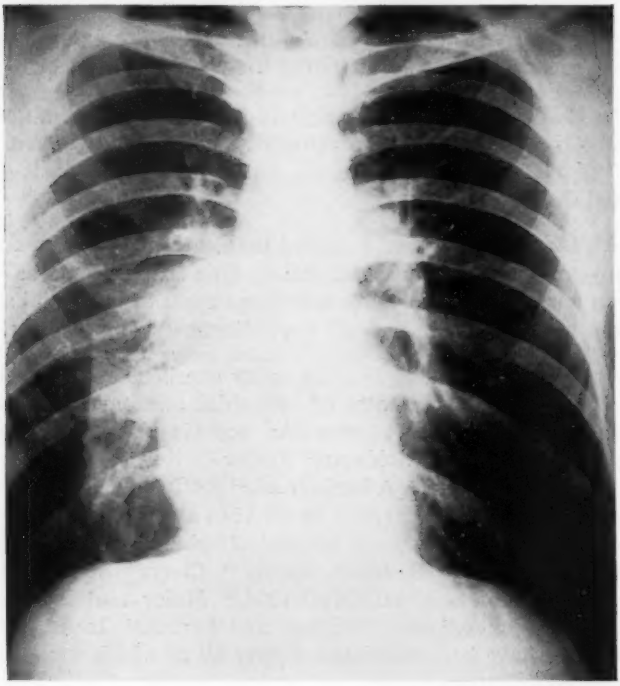
lignancy might occur. It would seem that adequate evidence is now available to merit a definite conclusion relative to the classification and treatment of this interesting neoplasm.

It has been stated that about 8 per cent of all primary bronchial tumors are adenomas. Clerf reported an incidence of

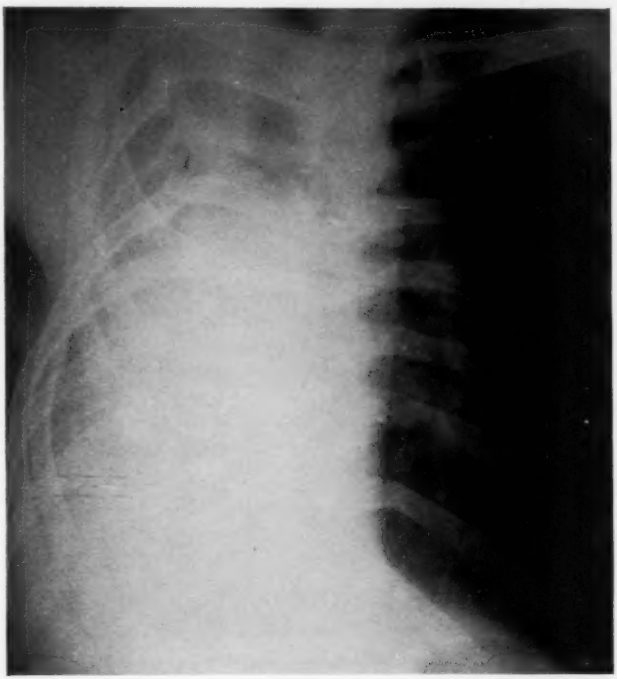


Case 1. Roentgenogram showing collapse of right lower and middle lobes which was secondary to adenoma in right main stem bronchus.

12 per cent of 278 neoplasms diagnosed bronchoscopically. Of 217 histologically proven primary lung tumors diagnosed at the Lahey Clinic since 1930, the incidence was 6.9 per cent, or only 15 adenomas. They reported nine of the cases within

the past three years, compared with six in the previous 15 years. This may indicate a more definite agreement on the histology.

Bronchial adenomas are more prevalent in the third and fourth decades. They are somewhat more prevalent in females.

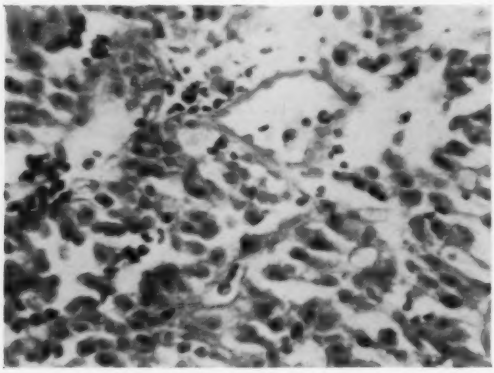


Case 1. Roentgenogram taken two years after right pneumonectomy for adenoma.

The tumor is usually located in the larger bronchi, hence readily visualized bronchoscopically; however, Maier and Fischer[19] have reported adenomas arising in small bronchi and not visible bronchoscopically.

One of the chief characteristics of the symptomatology of these growths is chronicity. Bronchial adenoma is a slow-growing tumor, hence has prolonged signs. Cough, wheeze and blood-tinged sputum may be the only symptoms for many months or even years; however, recurring bouts of fever and chest discomfort frequently diagnosed as atypical pneumonia by the clinicians and unresolved pneumonia by the Roentgenologists should arouse one's suspicion of an endobronchial new growth and be followed by a request for bronchoscopic examination. Further variations of symptoms depend on the endo- and peribronchial extension of the growth and the resulting mechanical conditions.

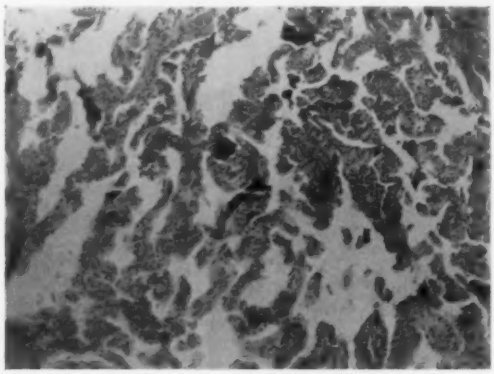
Two new cases are herein reported to further emphasize the protean nature the symptoms may present. These two cases had similar operative findings but very dissimilar preoperative histories.



Case 1. Photomicrograph showing glandular formation with incomplete lumen.

Case 1: A young man, aged 20, had six endoscopic treatments in five years of an adenoma in the right main bronchus. The tumor eventually became obstructive,atelectasis developed, and operation was necessary. At operation extensive hilar spread was found, and a pneumonectomy was indicated. Only four years have elapsed but he is in excellent physical condition. He is an instructor in a college of music, sings in the glee club and plays a wind instrument in the orchestra. Diagnosis was bronchial adenoma, carcinoid type.

Case 2: Mrs. H., age 32: no chest symptoms until six months before surgical treatment was instituted. Within one month she had two bouts of chills, fever and lower right chest discomfort. The diagnosis was atypical pneumonia. Roentgen diagnosis was unresolved pneumonia with question of a small area of segmental atelectasis of the right lower lobe. At bronchoscopy a small reddish new growth was seen in the bronchus of the posterior basal segment on the right side. Pathological diagnosis was bronchial adenoma, cylindroma type. Surgery revealed extensive peribronchial spread upward along the main stem, necessitating removal of the lower and middle lobes. The patient is well and asymptomatic after three years.



Case 2. Photomicrograph shows cylindroma type of cells with amyloid stroma.

It is interesting to note the various names used in the past to describe these tumors:

1931 — Geipel — Basal Cell Cancer.

1932 — Wessler and Rabin — Benign Bronchial Adenoma.

1935 — Kiernan — Carcinoid.

1935 — Moersch and Bowing — Adenocarcinoma.

1936 — Clerf and Crawford — Benign Glandular Tumor.

1937 — Zamora and Schuster — Vascular Adenoma.

1937 — Welt and Weinistein — Endothelioma.

1937 — Hamperl — Cylindroma and Carcinoid.

1938 — Womack and Graham — Mixed Tumor of the Lung.

1940 — Achsner — Reserve Cell Tumor.

1940 — Goldman — Polypoid Bronchial Tumor.

The type of cell is essentially the same in all bronchial adenomas, but there are distinct variations in the arrangement of cells which enable the tumors to be classified into two histological groups:

1. Consists of tubular and glandular alveoli.

2. Atypical structure and less degree of glandular formation, together with solid acini and irregular masses of cells. Mitoses are seldom seen. These variations in structure have caused confusion in the past and failure to recognize them has falsified diagnosis.

The most commonly used terms are Carcinoid and Cylindroma or Mixed Tumor. A large majority now reported are called Carcinoids. Pearson and Fitzgerald[22] state that all carcinoid tumors should be considered malignant. Argentaffine granules have been definitely demonstrated in only one bronchial adenoma (Holley[23]). This would indicate these bronchial tumors are not histologically identical with the carcinoids of the alimentary tract. There is a striking histologic similarity between bronchial carcinoids and the islet cell tumors of the pancreas.[9] Boyd[26] also noted a likeness between pancreatic islet cell tumors and carcinoids of the appendix.

According to Ewing[24] the term cylindroma may be used to designate any tumor in which the stroma appears in the form of elongated, twisted, thickened cords of hyaline substance. The term should be used only as descriptive and not implying any fundamental relation to the cylindromatous type of basal cell carcinoma.

The mixed tumor classification as suggested by Graham and Womack[13] is further supported by Holley[23] in his report of 39 cases of bronchial adenomas. Thirty-four were classed as carcinoid and five as mixed tumor; however, to add to the confusion, he states that the stroma in these five cases was not

of the myxomatous, osteoid or cartilaginous type usually associated with mixed tumors. To this extent the term mixed tumor is a misnomer.

The widest divergence of opinion occurs concerning these so-called mixed tumors or cylindromas. They have been considered as a bronchial tumor apart from the adenoma. They have a very close resemblance to the mixed tumor of the salivary glands and to basal cell carcinomas of the skin. Ewing[24] states that a marked similarity also frequently occurs between mixed tumors of the salivary glands and basal cell skin carcinomas.

Obviously the exact classification of these bronchial tumors is not yet determined. The relationship of these adenomas and alimentary carcinoids, pancreatic islet cell tumors, basal cell carcinomas of the skin and certain tumors of the salivary glands remains to be explained.

The malignant potentialities of bronchial adenomas offer an important problem. There has been a marked lack of agreement on this point, hence no uniformity of therapy. Such authorities as Foster-Carter[6] and Clerf[16] state that metastases do not occur and doubt if the tumors ever become malignant. Recently there have been several unequivocal reports of metastases. Anderson[8] reported metastasis to the liver. Laff and Neubuerger[9] reported an adenoma with cylindromatous features which metastasized to the opposite lung.

Adams *et al.*[25] presented five malignant bronchial adenomas. Moersch and McDonald[18] also reported five cases of definitely proved metastases. They noted that metastasis to the liver is followed by rapid development of hepatic insufficiency and death. Holley[23] reports two metastases to hilar lymph nodes and one to the liver. Metastases have appeared in the liver, bone, thyroid gland, skin, contralateral lung and regional lymph nodes.

SUMMARY AND CONCLUSIONS.

Bronchial adenoma is a potentially malignant tumor. The malignancy is low grade.

Most of the tumors occur in the larger bronchi. Mucous glands are present in bronchi 2 mm. in diameter, hence adenomas may occur in these smaller branches and not be visible bronchoscopically.

The tumors usually have an intact mucosa, which explains why cytologic study of bronchial secretions often gives negative results.

Exact histological classification has not been determined. Carcinoid and cylindroma (mixed tumor) are now the commonly used terms.

About 90 per cent of the cases can be diagnosed bronchoscopically; however, no one has presented histologic criteria by which, on biopsy, it can be predicted how much of the tumor is within the bronchial lumen or to what extent invasion or distal spread has occurred.

In suitable cases the most satisfactory results are obtained by surgical extirpation of the lesion. It may require bronchotomy, lobectomy or pneumonectomy. Patients of advanced years and those with the tumor at the carina may have to be treated endoscopically. A definitely pedunculated tumor may be removed by bronchoscopic means but the "iceberg" nature of these neoplasms must always be considered. What is seen may be only a small portion of a peribronchial involvement. Body section Roentgenography may be of valuable assistance.

Patients treated endoscopically must be re-examined repeatedly because of the tendency to recurrence. Severe hemorrhage may follow bronchoscopic removal and stenosis follow repeated coagulations.

The exact rôle of deep Roentgen therapy is not determined.

Even though many of these tumors do not fulfill our commonly accepted criteria for malignancy, their conduct is of such a malignant nature that total extirpation is the best form of treatment.

Two cases are reported which demonstrate this type of conduct: one of five years' duration and one of less than one year.

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PRIMER CONGRESO CENTRO AMERICANO DE OTO-RINO-LARINGOLOGIA.

The First Congress of Otorhinolaryngology for Central America will be held in San Salvador, El Salvador, Central America, Dec. 12-13, 1952. For further information contact Hector R. Silva, M.D., Secretary, Calle Arce 84, San Salvador.

A NEW DESIGN FOR A RONGEUR TO BE USED IN LOWERING THE BONY SEPTUM.*

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The removal of the nasal hump is one of the successes of plastic surgery which is frequently practiced, and the technique of this operation is familiar to all plastic surgeons.[1] The intranasal incision is made, and the overlying skin of the region is undermined, followed by elevating the periosteum over the parts to be removed. The excess tissue forming the hump is removed by cutting completely through at the desired level. The maxillary processes are fractured or separated by use of a saw, in order to allow their closer approximation over the bridge, to narrow the nose sufficiently.

When this last step is carried out, there is no need for recourse to the rules of geometry, to realize that pressing the maxillary processes toward the median line changes their relation to the perpendicular plate of the ethmoid, which forms the bony septum. The result is that the upper border of the septum forms a sharp ridge along the nasal dorsum in this region. The final result is that a dorsal line is created, which is covered by only a thin layer of skin. This condition lends itself easily to injury, by its tendency to act as a cutting edge. Thus the skin may be cut through by the bony ridge, leading to scar formation and more or less disfigurement.

Kazanjian[2] has devised a rongeur with which he removes the entire hump, without the use of the saw. In the hands of the writer, this method has not proved so efficient as the customary one, in which the hump is excised by using the saw. After the removal of the bony fragment, the remaining bony septum has usually been lowered by means of a rasp,

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or a common rongeur. The use of these instruments often leaves a rough, bony edge to form the dorsal line over the bridge.

To avoid this outcome of the operation, the writer has prepared a rongeur, with which the perpendicular plate can be lowered evenly and smoothly.

This instrument (see Fig. 1) is made of stainless steel, and is seven and one-eighth inches (20.5 cm.) in length. The

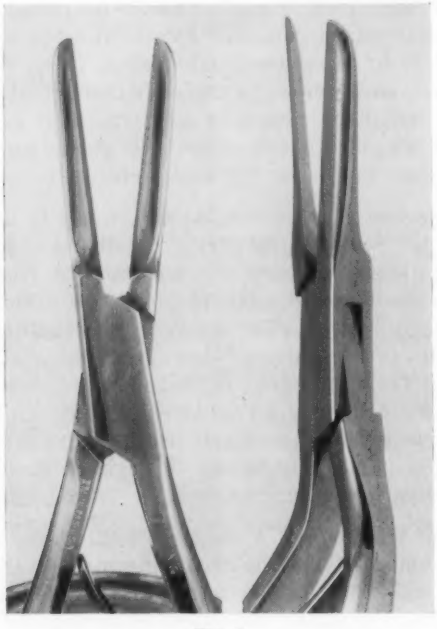

Fig. 1.

measurements at the hinge are one-half inch (1.2 cm.) by five-sixteenth inch (1.0 cm.). The blades are two inches (5.0 cm.) in length. They are beveled and are relatively thin. To allow for reaching the bony septum, they are canted to form an angle of 90 degrees between their upper surfaces. The

handles (shanks) are angulated to an obtuse degree, just behind the hinge, and are cross-hatched to provide a firm grip (see Fig. 2).

The special value of this instrument is that after the hump has been removed, the blades can be fitted between the cut

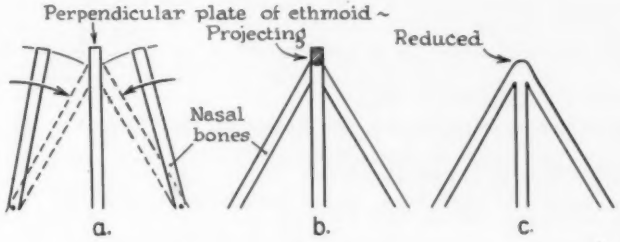

Fig. 2.

edges of the maxillary processes, to lie on either side of the bony septum, and the necessary amount of tissue can be cut from the septum accurately and easily. The anterior maxillary processes can then be pressed toward the median line, and the final presence of a roughened prominent dorsal ridge is avoided.

The writer has found that the use of this rongeur offers a particular advantage for narrowing the bridge in the operation for removal of a nasal hump.

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Manufacturer: Aurex Corp., 1117 N. Franklin St., Chcago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.

Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone T5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3.

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Rochester Model R-1; Rochester Model R-2.
Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model J-92; Silvertone Model P-15.
Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.
Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Vice-President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
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Meeting: Roosevelt Hotel, New Orleans, La., May 1-2, 1953.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Roosevelt Hotel, New Orleans, La., Apr. 26-27, 1953.

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Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Roosevelt Hotel, New Orleans, La., April 28-29, 1953 (mornings only).

MID-WINTER.

Triological Council Meeting—Hotel Syracuse, Syracuse, N. Y., 9 a.m., Jan. 6, 1953.

Eastern Section Meeting—Hotel Syracuse, Syracuse, N. Y., Jan. 7, 1953.

Southern Section Meeting—Andrew Jackson Hotel, Nashville, Tenn., Jan. 12, 1953.

Middle Section Meeting—The Drake, Chicago, Ill., Jan. 19, 1953.

Western Section Meeting—The Elks Club, Los Angeles, Calif., Jan. 24, 1953.

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Meeting:

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Meeting: Palmer House, Chicago, Ill., Oct. 6-10, 1952.
Meeting: Roosevelt Hotel, New Orleans, La., Apr. 21-25, 1953.

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Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.
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Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
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Place: Army and Navy Club, Washington, D. C.

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Meeting: Edgewater Gulf Hotel, Edgewater, Miss., May 11, 1953.

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Meeting:

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Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonzo, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: January, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

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For Otolaryngology, Charlottesville, Va., Nov. 21-22, 1952.
Spring Meeting: Hot Springs, Va., May, 1953.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Recording Secretary, E. N. T. Section: Dr. Donald B. Hull.
Chairman of Eye Section: Dr. Deane Hartman.
Recording Secretary, Eye Section: Dr. Robert Norene.
Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

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Secretary of Assemblies: Dr. Cesar Rodriquez.
Time and Place: Caracas, Venezuela, July 31, 1954.

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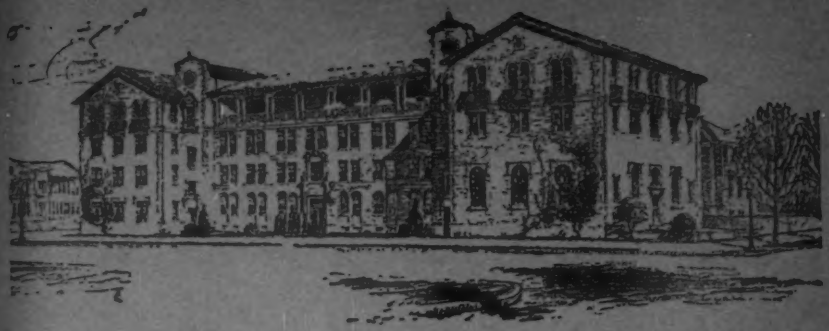

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